HEMISPHERIC DIFFERENCES IN EMOTIONAL PSYCHOPHYSIOLOGY

By

BETH S. SLOMINE

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Beth S. Slomine

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Chairperson: Dawn Bowers
Cochairperson: Russell M. Bauer
Major Department: Clinical & Health Psychology

Two theories have been proposed to explain the organization of emotions within the cortical hemispheres. According to the global right hemisphere model, the right hemisphere takes a predominant role in modulating emotions. Based on the global theory, patients with right hemisphere damage (RHD) have a deficit in emotional processing of all emotions. According to the other hemispheric theory of emotion, the bivalent model, the right hemisphere modulates negative emotions and the left hemisphere modulates positive emotions. This model predicts that RHD patients would be deficient in emotional processing of negative emotions, whereas patients with left hemisphere damage (LHD) would be impaired in processing positive emotions.

In this study, emotional experience as measured by autonomic responding, facial muscle activity, and verbal report was examined in 12 patients with RHD, 12 patients with LHD, and 24 normal control subjects (NCS) during anticipation of shock and reward. Results revealed that during the shock condition, RHD subjects displayed a deficit in skin conductance responding compared with the NCS, but not compared with the LHD subjects. None of autonomic or facial muscle variables differentiated the reward from the control condition during the reward task. These results are discussed in light of the global and bivalent theories of emotion as well as neuroanatomic correlates of electrodermal activity.

CHAPTER 1
REVIEW OF THE LITERATURE

Introduction

Patients with unilateral brain damage have been used to investigate hemispheric contribution to emotional perception, voluntary expression, and to a lesser extent "experience" as indirectly assessed through physiologic arousal, overt behavior, and verbal report. Although some studies have suggested that differences in post-stroke mood occur following right hemisphere damage (RHD) and left hemisphere damage (LHD), few studies have assessed brief emotional experience while measuring psychophysiological and behavioral indices of emotion in these patients. Moreover, when emotional experience has been studied using physiological indices of emotion, patients needed to decode emotional stimuli, which may be problematic for some RHD patients. Additionally, no study to date has employed facial EMG when examining emotional experience in unilaterally damaged patients. In the current project, stroke patients with left or right hemisphere lesions participated in two experiments designed to examine specific deficits in pleasant and unpleasant emotional experience as a function of unilateral brain damage. Both physiological

responding, facial behavior, and verbal report were measured during "in vivo" affective situations.

Before discussing the experiments further, a brief overview of the literature is provided. The review includes prominent theories of emotion which have stemmed from the works of James (1884/1922), Lange (1922), and Cannon (1927). Moreover, theories of hemispheric specialization of emotion are provided. Specifically, two predominant neuropsychological theories of emotion are explored: (1) the global right hemisphere theory which states that the right hemisphere is responsible for affective processing; and (2) the bivalent view which conceptualizes the right hemisphere as predominant for negative emotions and the left hemisphere as predominant for positive emotions. In addition, studies of hemispheric differences in emotional evaluation, expression, arousal, and mood are discussed. Lastly, an overview of emotional psychophysiology is presented.

Theories of Emotion

The quest to understand emotion has stimulated the development of many theories and much empirical data over the past century. According to Kleinginna and Kleinginna (1981) the numerous definitions of emotion complicate research in emotion. After an extensive review of emotional definitions, they classified psychological definitions of emotions into 11 non-mutually exclusive categories on the

basis of emotional phenomenon and theoretical issues. They concluded that a definition of emotion should be broad enough to include significant aspects of emotion, but still be able to distinguish emotion from other psychological phenomenon. They suggested the following definition:

> Emotion is a complex set of interactions among subjective and objective factors, mediated by neural/hormonal systems, which can (a) give rise to affective experiences such as feelings of arousal, pleasure/displeasure; (b) generate cognitive processes such as emotionally relevant perceptual effects, appraisals, labeling processes; (c) activate widespread physiological adjustments to the arousing conditions; and (d) lead to behavior that is often, but not always, expressive, goal directed, and adaptive. (p. 355)

Like the numerous definitions of emotions, there are many theories of emotion. These differ in their conceptualization of emotional experience and the role of cognition in emotional experience. A few prominent emotion theories are described below.

James-Lange versus Cannon Debate

James (1884/1922) and Lange (1922) were the first to challenge the common sense view that perception of an event was followed by the experience of emotion. James stated that "...the bodily changes follow the perception of the exciting fact, and that our feelings of the same changes as they occur is the emotion" (p.13). James proposed that, in order to experience emotion, one must simultaneously exhibit physiological and expressive changes, such as tensed muscles and quickened heart rate during fear. Specifically, the James-Lange theory states that perception occurs when an

object stimulates one or more sense organs relaying afferent impulses into the cortex. Next, cortical efferents send information to skeletal and visceral musculature producing complex changes. Lastly, sensory information from the affected musculature is projected back to the cortex. Perception of this sensory information produces the experience of emotion. In the early 20th century, the James-Lange theory predominated the study of emotion (Izard, 1977).

In 1927, Cannon presented five criticisms of James-Lange's hypotheses that perception of autonomic/visceral changes are responsible for the experience of emotion. First, Cannon cited evidence that spinal cord transections in dogs, in which the sensations of the viscera were separated from the CNS, did not alter emotional experience. Additionally, he stated that cats who had their entire sympathetic division of the autonomic nervous system removed showed all the manifestations of rage when presented with a dog (i.e., hissing, growling, and retraction of the ears) except the cats did not raise the hairs on their backs. Second, Cannon pointed out that the same visceral changes occur during sympathetic arousal even though different emotion states may be experienced. Additionally, sympathetic arousal produces similar changes in non-emotional states such as fever or exposure to cold. Third, Cannon argued that the viscera are relatively insensitive

structures and changes are often not experienced consciously. Fourth, he stated that visceral changes are slow and thus, cannot be a source of emotion. Fifth, he claimed that producing artificial visceral changes does not produce affect. He used adrenalin as an example stating that adrenalin produces bodily changes that are not accompanied by affective states. He concluded that the sensation of visceral responses cannot produce affect.

Cannon hypothesized that "emotional expression results from action of subcortical centers" (p.115). Cannon cited studies in which various types of decorticate animals displayed abnormal affective responses, whereas animals with hypothalamotomies failed to display affective behavior. Consequently, Cannon concluded that the cerebral cortex normally inhibits thalamic activation. He purported that during normal emotional experience sensory information arrives at the cortex and is projected to the hypothalamus releasing it from cortical control. Cannon proposed that hypothalamic activation relays information to somatic musculature and smooth musculature of the viscera to produce characteristic manifestations of emotion. Simultaneously, the hypothalamus projects to cortex which produces the conscious awareness of emotion. According to Cannon muscular changes, visceral changes, and conscious experience of emotion all occur simultaneously. The result is intense

emotional experience accompanied by behavior and physiological indices of emotion.

Later scientists elaborated on Cannon's theory. Papez (1937) postulated that a circuit of emotion exists that relays information to the hypothalamus from the anterior thalamus, cingulate cortex, and hippocampus. He posited that emotion originates in the hippocampal formation and is relayed through the above circuit to the cortex. He described the cingulate gyrus as the receptive cortical region for emotion. About a decade later, MacLean (1949, 1952) described the limbic system as a group of phylogenetically old cortical structures that are involved in emotion.

More recently, LeDoux (1989) has argued that emotion and cognition are mediated by separate though interacting neural systems. According to LeDoux, the amygdala is the major component of the brain's affective processing system, whereas the hippocampus is critically involved in cognitive processing. Both affective and cognitive computations can occur without conscious awareness. According to LeDoux, affective computations occur via thalamo-amygdala projections which process the affective significance of simple sensory cues, whereas the cortico-amygdala pathway processes complex affective stimuli. The thalamo-amygdala projections are adaptive because this pathway often leads directly to motor responses with brief processing time,

i.e., fleeing from a dangerous snake. LeDoux proposed that the amygdala receives exteroceptive sensory, interoceptive sensory, and neural input. In addition, LeDoux (1984) explains that sensory information from the peripheral nervous system feeds back to the amygdala to intensify amygdala excitation and increase the duration and intensity of the experience of emotion.

LeDoux suggested that the amygdala performs the functions that Cannon (1927) and Papez (1937) thought belonged to the hypothalamus. Together, Cannon, Papez, and LeDoux challenged the James-Lange Theory in hypothesizing that emotional experience can be generalized in the brain without the participation of the peripheral nervous system. However, none of these theories discuss the differing roles that the right and left cerebral hemispheres may play in modulating emotional behavior.

Appraisal Theories

Other theorists have attempted to address Cannon's criticism of autonomic feedback proposed by James and Lange. Russell (1927/1961) stated that cognition as well as physiological feedback compose the experience of emotion. Within the past few decades, some theorists have viewed emotion as a phenomenon developing from cognitive appraisal of an event, situation, or condition. Arnold (1960) described emotion as the nonrational judgement of an object which follows perception and appraisal. Schacter and Singer

(1962) proposed that physiological arousal along with cognitive appraisal are both essential for emotion to result. They suggested that some event or condition creates physiological arousal which is combined with evaluation of the event or condition (cognitive appraisal) to lead to the experience of emotion.

Central to appraisal theories is the view that the experience of any emotion (i.e., joy, anger, fear) involves the same physiological arousal, but different cognitive appraisals. Lazarus and Averill (1972) explained that emotion results from appraisal of stimuli and the formulation of a response. In their view, appraisal reduces and organizes stimulus input to a specific concept, (e.g., a threat). Lazarus and Averill also asserted that personal psychological structure and social norms also influence appraisal. Most importantly, they concluded that appraisal determines the specific emotional experience. For example, anger has been associated with the perception of goal obstacles, whereas fear is associated with perceived uncertainty about and unpleasant situation (Ellsworth & Smith, 1988). However, these theorists place little or no emphasis on neural hardware which might underlie or contribute to appraisal.

Differential Emotion Theory

The Differential Emotion Theory was developed by Tomkins (1962, 1963) who proposed that awareness of

proprioceptive feedback from facial muscles constitutes the experience of emotion. According to Tomkins, emotion-specific innate programs for groups of facial expressions are stored in subcortical centers. Tomkins hypothesized that once an emotion has been activated, facial feedback is provided to the cortex. Additionally, Tomkins argued that it is the facial feedback that initiates visceral activation.

Differing slightly from Tomkins, Izard (1977) argued that emotion involves three components; neural activity or the density of neural firing per unit time, striate muscle feedback to the brain, and subjective experience. Izard posited that each component can be dissociated from the others, but that the three are normally interdependent. Specifically, according to Izard, internal or external stimuli affect the gradient of neural stimulation in the limbic system and sensory cortex. Information from these areas are relayed to the hypothalamus which plays a role in determining the facial expression to be effected. From the hypothalamus, impulses are relayed to the basal ganglia where the neural message for facial expression is mediated by motor cortex. Impulses from motor cortex, via cranial nerve VII lead to the specific facial expression. Cranial nerve V receives sensory input from the face and projects, via the posterior hypothalamus, to sensory cortex. It is

the cortical integration of facial expression feedback that generates subjective experience of emotion.

Proponents of the Differential Emotion Theory have conceptualized a certain number of fundamental emotion categories which are comprised of specific phenomenological characteristics, expressive responses, and physiological patterns. Darwin (1872) was one of the first to discuss his observations of the expression of discrete emotions. He described many emotions which he viewed as having corresponding facial expressions which are universally displayed and recognized by humans cross culturally. According to Izard (1977) there are 10 fundamental emotions such as happiness, sadness, anger, fear, and disgust.

The concept of discrete emotions developed mostly from direct observation and study of facial expressions. Fridlund, Ekman, and Oster (1987) reviewed the literature on facial expressions including phylogenetic, cross-cultural, and developmental research. They determined that there is much support for discrete emotions. Their conclusions, based on the literature, are as follows: (1) phylogenetic studies have shown that many nonhuman primates show a variety of differentiated facial patterns and similar facial patterns have been observed among human and nonhuman primates; (2) cross-cultural studies have revealed that members of different cultures display the same facial expressions and use analogous emotion labels when

identifying the underlying emotions of posed expressions; and (3) developmental research has indicated that facial musculature is fully formed and functional at birth and infants display many facial expressions similar to adult expressions. Also, infants demonstrate differential responses to facial expressions by 3 months of age and have the capacity to imitate facial movements within the first few days of life.

One problem not addressed by the differential emotions theorists is whether spontaneous experience of these emotions is accompanied by the occurrence of the predicted facial expression (Davidson, in press). For instance, Davidson stated that little is known about the incidence of different facial expressions depending on context or type of emotion elicitor (i.e., imagery, emotional film clip). For example, Tomarken and Davidson (1992) found very few overt expressions of fear in response to fear film clips. Also, Davidson (in press) raised questions concerning the facial expressions of positive emotion. Specifically, he indicated that while there are multiple forms of positive affect as evidenced using behavioral, subjective, and physiological indices, there is only one facial expression indicative of the experience of positive emotion.

Dimensional Approaches

In an attempt to explain the polarity of emotion, dimensional theorists have conceptualized emotion as varying

on two or three polar dimensions. Wundt (1896) suggested that emotions can be conceptualized in terms of three different dimensions: pleasantness-unpleasantness, relaxation-tension, and calm-excitement. In addition, the dimensional views of emotion were supported by Cannon's (1927) claim that the same visceral changes occur in different emotional states. Consequently, theorists such as Duffy (1957) conceptualized emotions as varying along a general state of activation or arousal. Other contemporary investigators have used dimensions to characterize facial expressions (e.g., Scholsberg, 1941; Osgood, 1966) and verbal report (e.g., Russell & Mehrabian, 1977). Lang (1985) stated that most variance within factor analytic studies of the verbal report of emotional experience was accounted for by two dimensions, activation (arousal-quiescence) and valence (pleasure-displeasure). Because the bidimensional view seems to neglect a certain amount of variance, Lang proposed that the dimension termed dominance-submission by Russell and Mehrabian (1977) may account for the residual variance.

Similar to the view of the discrete emotions theorists, Lang (1985) suggested that emotional behavior has developed phylogenetically for basic survival tasks (e.g., searching for food or fighting for territory). Further, Lang hypothesized that the combination of valence (approach vs. avoid), arousal (energy mobilization), and dominance

(postural stance) are critically important for smooth execution of behaviors necessary for success in survival tasks. Lang asserted that it is essential to determine how emotion is represented in memory in order to ascertain how emotion drives cognitive processing. Lang proposed that emotion information is coded within memory in the form of propositions which are organized into associative networks. The associative networks are comprised of three tiers; semantic codes, stimulus representation, and response programs.

According to Lang's Bioinformational Theory (1979, 1984), emotions are associated with action. Access of emotional propositions are associated with efferent outflow, and thus emotion can be measured in terms of three response systems; verbal report, overt behavior (i.e, facial expression, body posturing, and emotional prosody), and peripheral and central physiological measures. However, only stable networks which are called emotion prototypes, such as those found in phobics, demonstrate a reliable behavioral output in all three response systems. Consequently, emotional experience is an epiphenomenon of the 3 response systems which reflect an underlying centrally represented propositional network.

Taken together, theories of emotion differ quite dramatically in their emphasis on and definition of emotional experience. James and Lange view emotional

experience as the awareness of bodily sensations associated with emotion. Cannon, on the other hand, views conscious awareness of emotion as arising from neurological activation which may be accompanied by visceral and muscular changes. Papez, MacLean, and LeDoux support this view. Appraisal theorists emphasize the importance of cognition combined with physiological arousal in the awareness of emotion. Discrete emotion theories view the experience of categorical emotions which corresponded to specific facial expressions. Lastly, most dimensional theorists emphasize the experience of emotion based on two or three polar emotional dimensions, whereas Lang views emotional experience as an epiphenomenon of overt behavior, physiological activity, and verbal report.

For purposes of the present study, emotional experience is defined as a psychological phenomenon or subjective experience which can be measured indirectly through physiological measures, verbal report, and overt behaviors (e.g., facial muscle responses). Because emotional experience is not directly observable, problems are inherent in any definition of and attempt to measure it. In terms of the present definition of emotional experience, it is unclear what the impact of decreased responding in any of the three response systems means in terms of emotional experience. For instance, if an individual reports experiencing anxiety, but displays no physiological or overt

responses, it is unknown whether there is a disconnection between experience and motor output or whether the person is not experiencing the emotion as completely as someone who reacted with all three response systems.

Hemispheric Asymmetry of Emotion

Along with general psychological theories of emotion, investigators have examined the organization of emotion in the brain. Historically, emotion has been associated with the limbic system (Papez, 1937; MacLean, 1952). More recently, neuropsychologists have examined the role of the cerebral hemispheres in modulating emotional behavior. Research involving neurologically impaired patients has aided in developing an understanding of how various domains of emotional behavior (i.e., evaluation, expression, arousal) are disrupted by focal lesions of the left and right hemispheres. Based on some clinical studies, along with findings from normal individuals (see review, Heilman, Bowers, & Valenstein in press), inferences have been made regarding the neural networks that might underlie different aspects of emotional behavior including evaluation, expression, arousal, and experience.

Early observations of individuals following hemispheric damage revealed differences in mood reactions depending on whether the left or right hemisphere was involved. Babinski (1914) was one of the first to note that patients with right hemisphere damage (RHD) appeared indifferent or

euphoric. Others have reported similar observations (Denny-Brown, Meyer, & Horenstein, 1952). Denny-Brown et al. described a 55 year old woman with a right parietal infarct, who appeared "indifferent" towards her illness as well as apathetic towards her family's affairs. By contrast, individuals with left hemisphere dysfunction (LHD) have been observed to appear depressed, which was termed "catastrophic reaction" by Goldstein (1948). Terzian (1964) noted that injection of sodium amytal into the left carotid artery, which inactivated the left hemisphere, was associated with a depressive reaction, whereas injection of sodium amytal into the right carotid artery was associated with an euphoric reaction. More systematic large-scale studies of RHD and LHD patients have been consistent with the early clinical reports. Gainotti (1972) investigated the verbal expressions and behavior of 160 patients with left and right hemisphere lesions. Behaviors indicative of catastrophic reactions or anxious-depressive mood were more frequent among LHD patients, while indifference reactions were more prevalent among RHD patients. Observations of post-stroke mood changes has generated a large body of research over the past 20 years in an attempt to understand the contributions of the left and right hemispheres to emotion.

Two prominent theories of hemispheric differences in emotion have arisen from the clinical studies reported above. According to the global right hemisphere view, the

right hemisphere is involved in interpreting emotional stimuli and has a unique relationship to subcortical structures which mediate cerebral arousal and activation (e.g., Heilman, Watson, & Bowers, 1983). Consequently, damage in the right hemisphere interferes with processing emotional stimuli, programs of expressive behavior, and cerebral arousal and activation. In contrast, the bivalent view of emotion posits that the anterior portion of the right hemisphere is dominant for negative/avoidance emotions and the anterior region of the left hemisphere is dominant for positive/approach emotions (e.g., Fox & Davidson, 1984). According to the bivalent view, right hemisphere damage causes positive/approach affect and left hemisphere damage evokes negative/avoidance affect. Both models and the empirical research in support of each are discussed below.

Global Theory of Emotion

According to the global right hemisphere model, observations of emotional indifference in RHD patients can be explained by the right hemisphere's specialization for coding nonverbal affective signals and mediating arousal and activation (Heilman et al., 1983). The global right hemisphere theory is supported by research exploring emotional evaluation, expression, and arousal/activation, which has revealed that RHD patients are deficient in interpretation of emotional stimuli, are emotionally

flattened, and physiologically hypoaroused. The relevant research is discussed below.

Evaluation of emotion

Most of the research in support of the global right hemisphere view of emotion has arisen from investigations of evaluation and perception of affective stimuli (i.e., facial expression and emotional prosody). Many patients with RHD have impairments in identifying and discriminating facial expressions. This research was initially conducted by DeKosky, Heilman, Bowers, and Valenstein (1980) and has been consistently replicated across other laboratories (Cicone, Wapner, & Gardner, 1980; Etcoff, 1984; Bowers, Bauer, Coslett, & Heilman, 1985). From an historical perspective, one critical issue was whether the RH superiority in identifying facial expressions was secondary to the role of the RH in mediating complex visual configurational stimuli. Evidence against this view point comes from covariance studies, individual case reports, and studies which find RHD patients impaired in identifying facial affect when it has been verbally described.

First, in covariance studies, visuoperceptual ability has been controlled for and equated statistically. In these studies, deficits in RHD patients in recognition of affective facial expressions have been observed above and beyond deficits in visuoperceptual ability (Ley and Byrden, 1979; Bowers et al., 1985). Second, case descriptions have

documented dissociations between performance on visuoperceptual facial recognition and performance on affective facial expression recognition (Dekosky et al., 1980). Third, Blonder et al. (1992) found that RHD patients were impaired relative to LHD patients and NHD controls in identifying emotion associated with a verbal description of a non-verbal signal, i.e., he scowled. Similar results were found in RHD patients compared to LHD patients and normal controls when asked to imagine facial expressions (Bowers, Blonder, Feinberg, & Heilman, 1991). Because these nonverbal affect signals were verbally described, poor performance of the RHD group could not be attributed to perceptual impairment.

Taken together, these studies suggest that there are specific subsystems for processing affective facial stimuli. This evidence is comparable to findings in the animal literature. Using single cell recordings, neuroscientists have identified visual neurons in the temporal cortex and amygdala of monkeys that responded selectively to faces and to facial expressions (Perret et al., 1984; Leonard, Rolls, & Wilson, 1985).

In addition to deficits in comprehension of emotional faces, many patients with RHD also have impairments in understanding emotional prosody. For example, many patients with RHD have difficulty identifying emotional prosody, which includes the pitch, tempo and rhythm of speech.

Discrimination of affectively intoned speech was found to be worse in patients with RHD in the temporoparietal regions compared to patients with LHD (Tucker, Watson, & Heilman, 1977; Heilman, Scholes, & Watson, 1975; Ross, 1981).

In addition, there is evidence to suggest that RHD patients are impaired in understanding nonemotional as well as emotional prosody (Weintraub, Mesulam, & Kramer, 1981). Both RHD and LHD patients were impaired compared to NHD controls in nonemotional prosody, while RHD were more impaired than the LHD patients in emotional prosody (Heilman, Bowers, Speedie, & Coslett, 1984). Consequently, these authors conclude that both hemispheres are important in comprehension of nonemotional prosody, but the right hemisphere plays a more vital role in the comprehension of emotional prosody.

Not all studies find hemispheric specific prosody dysfunction. Schlanger, Schlanger, and Gerstmann (1976) found no differences between RHD and LHD patients in comprehension of emotional prosody; however, only 3 of 20 RHD patients in this study had temporoparietal lesions. More recently, Van Lancker and Sidtis (1992) found equally poor affective prosodic recognition in RHD and LHD patients. Moreover, they determined that LHD and RHD patients use different cues in attempting to recognize affective prosody. Specifically, RHD patients tended to use timing cues, whereas LHD patients used information about pitch. These

authors concluded that affective prosody is a multifaceted process which cannot simply be explained by differences in hemispheric specialization.

Studies of normals using dichotic listening tasks have also been employed to explore hemispheric differences in processing emotional prosody. In dichotic listening, two different messages are simultaneously presented to the right and left ears. Words were recalled best from the right ear indicative of left hemisphere superiority (Kimura, 1967), while mood of the speaker was recalled better from the left ear, suggestive of right hemisphere superiority in processing emotional prosody (Haggard & Parkinson, 1971; Ley & Bryden, 1982).

In contrast to the tasks involving nonverbal signals, evidence for a unique role of the right hemisphere in mediating emotional understanding of messages that are conveyed through propositional language is equivocal. Recognition of emotional words has been found to be better when presented tachistoscopically to the right hemisphere (Graves, Landis, & Goodglass, 1981). However, RHD and LHD patients did not differ in the ability to comprehend the meaning of emotional and nonemotional words (Morris et al., 1992), the ability to identify emotionality of short propositional sentences (Heilman et al., 1984; Cicone, Wapner, & Gardner, 1980; Blonder, Bowers, & Heilman, 1991),

or the ability to judge similarity between two emotional words (Etcoff, 1984).

However, recent evidence contradicts these findings. Borod et al. (1992) found that, when compared to LHD and NHD patients, RHD patients were more impaired in identifying and discriminating emotional words and sentences. In addition, RHD patients were impaired in their understanding of emotionality in complex narratives (Gardner, Brownell, Wapner, & Michelon, 1983; Gardner, Ling, Flam, & Silverman, 1975; Brownell, Michelon, Powelson, & Gardner, 1983). The deficits of RHD patients in understanding complex narratives may not be related to emotion, but to difficulties of RHD patients in drawing inferences, reasoning, and interpreting figures of speech (Heilman, Bowers, & Valenstein, in press). However, this explanation does not explain the results of Borod et al. (1992) who found that RHD were impaired in identifying and discriminating words and short sentences.

Taken together, the above studies indicate that patients with RHD have more difficulty than LHD patients and NHD controls in evaluating nonverbal signals of emotion, including facial expressions, emotional prosody, and verbal messages of emotions. Moreover, RHD patients are equally impaired for both positive and negative emotional signals. Although some deficits in recognition of facial expressions in RHD patients are related to general dysfunction in visuospatial ability, others are apparently independent of

visuospatial ability. In part, some deficits in affective prosody may be due to more elemental dysfunction in complex auditory analysis. In contrast to nonverbal affective signals, the role of the right hemisphere in processing verbal emotional signals remains unclear. At present, some argue that an emotional semantic network is widely distributed between the hemispheres whereas other argue that the RH may be dominant for emotional semantics.

Expression of emotion

The global right hemisphere view of emotion has also been supported by investigations of deficits in expression of emotion. Overall facial expressivity of emotions has been evaluated in RHD, LHD, and NH controls. Some authors have reported that RHD patients were less spontaneously expressive than LHD and NH controls (Blonder, et al., 1991; Borod, Koff, Lorch, & Nicholas, 1985; Borod, Koff, Perlman-Lorch, & Nicholas, 1988; Buck & Duffy, 1980). However, Weddell, Miller, and Trevarthen (1990) found LHD and RHD patients who had tumors were equally impaired and less expressive than NHD controls. When excisions occurred or tumor and CVA patients were combined, RHD and LHD patients did not significantly differ from controls (Kolb & Milner, 1981; Mammacuri, et al., 1988). Additionally, re ent evidence exists from studies using a carefully delineated facial scoring system which contradicts the findings that RHD patients are less facially expressive. For example, no

differences in facial expressiveness has been found between LHD and RHD patients when Ekman's facial action scoring system (FACS) has been used (Mammacuri, et al., 1988; Caltagirone, et al., 1989).

Other studies have examined the ability of RHD and LHD patients to voluntarily pose specific facial expression. Some investigators have found that RHD patients were more impaired than LHD patients and NHD controls in their voluntary expression of facial affect (Borod, Koff, Perlman-Lorch, & Nicholas, 1986; Borod, & Koff, 1990; Kent, et al., 1988; Richardson, Bowers, Eyeler, & Heilman, 1992). Other investigators (Kolb and Taylor, 1990) found that RHD and LHD patients are equally impaired relative to NHD controls, whereas others found no differences in expressivity among these three groups (Caltagirone et al., 1988; Heilman et al., 1983; Weddell, et al., 1990).

Borod (submitted) reviewed the literature on facial expressiveness in unilateral damaged patients. She concluded that the patients in those studies finding RHD patients to be more impaired than LHD and normal controls differed from those in which differences were not found. Specifically, she noted that the first group was more likely to be older, male, with cerebrovascular pathology, and a longer time since disease onset. The second group was more likely to have tumor pathology. Additionally, subjective ratings were used in the first group, while FACS and

concealed videotapes were used in the second group. One problem with these differences is that stroke patients may have more severe cognitive deficits than comparable tumor patients (Anderson, Damasio & Tranel, 1990). Secondly, acute pathology is associated with more pervasive deficits (Borod, in press). Thirdly, FACS may be insufficiently sensitive to facial expressive communication (Buck, 1990).

Asymmetries in facial expressiveness have also been examined in normal adults. In a recent review of 23 studies of spontaneous expression and 24 studies of posed expression, Borod (in press) concluded that the left hemiface is more intense and moves more than the right hemiface. According to Borod, these results were stronger for negative than positive emotions. There have been fewer studies of prosodic emotion than facial expression of emotion in patients with unilateral damage. Studies of spontaneous prosodic expression have revealed deficits in RHD patients compared to LHD patients and NHD controls (Ross & Mesulam, 1979; Borod et al., 1985; Gorelick & Ross, 1987; Ross, 1981). Similar results were found in investigations of voluntary affective prosody, such that RHD patients showed impairment relative to LHD and NHD controls (Borod et al., 1990; Gorelick & Ross, 1987; Tucker, et al., 1977). However, Cancelliere and Kertesz (1990) found no impairments in either RHD and LHD patients relative to NHD controls.

Emotional arousal/activation

Few studies have examined affective psychophysiological reactivity in brain-lesioned individuals. In the most commonly used procedure, emotional slides have been used to evoke affective responses while skin conductance response (SCR) is measured. Findings indicate that normals and patients with LHD have significantly higher SCRs to emotional than neutral slides. In contrast, RHD patients do not differentially respond to emotional and neutral slides (Morrow, Vrtunski, Kim, & Boller, 1981; Zoccolatti, Scabini, & Violani, 1982).

Similar results were obtained by Meadows and Kaplan (1992) using slides depicting neutral and negative content (i.e., mutilations). Relative to NHD controls, RHD patients had smaller SCRs to both emotional and neutral slides, LHD patients had higher SCRs to both types of slides. Contrary to the above findings, Schrandt, Tranel, and Damasio (1989) found that left hemisphere lesions and many right hemisphere lesions did not interfere with SCR during presentation of emotional slides. In this study, patients with focal lesions in left or right frontal, parietal, or temporal lobes were examined. Only patients with right hemisphere lesions involving the supramarginal gyrus displayed abnormal SCRs.

In another study, Heilman, Schwartz, andWatson (1978) investigated SCR while a mildly noxious electrical stimulus

was delivered to the forearm ipsilateral to the lesion in RHD, LHD, and NH patients. The RHD group had smaller SCRs than either the LHD or NH groups. Also, the LHD group had a higher SCR than the normal group.

Cardiovascular activity has also been examined in patients with LHD and RHD. Yokoyama, Jennings, Ackles, Hood, and Boller (1987) examined RHD, LHD, and NC patients using a reaction time task, while HR interbeat intervals were obtained. The controls and LHD subjects displayed anticipatory deceleration, followed by postresponse acceleration. The HR responding of the RHD patients varied little during anticipation and postresponse.

To sum, emotional slides evoke smaller SCR or less arousal, in right hemisphere damaged patients compared to NHD and LHD patients. Moreover, one study only found this distinction in patients with right parietal lesions. Additionally, in some studies, LHD patients responded with accentuated SCRs, (i.e., greater arousal), in response to emotional slides. Similar findings of decreased SCRs in RHD patients and increased SCR in LHD patients have been obtained in response to mildly noxious stimuli. Also, patients with RHD have attenuated HR reactivity in response to a reaction time task. Taken together, it appears that RHD patients are hypoaroused and LHD patients may be hyperaroused in response to emotional, painful, or attention-demanding stimuli.

Bivalent Model of Emotion

In its simplest form, the bivalent model posits that the right hemisphere is specialized for negative/avoidance emotions, whereas the left hemisphere is specialized for positive/approach emotions. According to the bivalent model, the catastrophic reaction noted in left hemisphere patients results from the predominance of the right hemisphere's negative emotion. On the other hand, the observations that right hemisphere damaged patients are euphoric or cheerful can be explained by the overcontrol of the left hemisphere's mediation of positive emotions (Davidson & Fox, 1982; Kinsbourne & Bemporad, 1984; Reuter-Lorenz & Davidson, 1981). This model was first based on observation of emotional behavior during inactivation of the left and right hemispheres with injection of sodium amytal (Terzian, 1964).

Evaluation of emotion

Research investigating the hemispheric differences during evaluation of nonverbal signals of emotion has yielded conflicting results. Although tachistoscopic studies in normals generally support the view that the right hemisphere is superior for processing emotional faces (i.e., Suberi & McKeever, 1977), closer examination reveals some support for the bivalent view of emotion. For example, the finding of right hemisphere superiority was attenuated with happy and angry facial expressions, which can be

conceptualized as approach emotions (Suberi & McKeever, 1977). Additionally, Reuter-Lorenz and Davidson (1981) presented subjects with an emotional face and a neutral face of the same individual simultaneously to each visual field. Reaction times for identifying happy expressions were faster during presentation to the right visual field (left hemisphere) and faster for sad expressions when presented to the left visual field (right hemisphere). However results have not been consistently replicated (Duda & Brown, 1984; McLaren & Bryson, 1987), and the vast majority of studies of affect perception in normals or focal lesion patients failed to demonstrate hemisphere-specific valence asymmetries.

Expression of emotion

Many studies of facial expressiveness have found that the left side of the face is more expressive than the right. These studies have been interpreted as reflecting a dominant role of the right hemisphere in emotional expression (Sackeim & Gur, 1978; Borod, Koff, & White, 1983; Campbell, 1978; Heller & Levy, 1981; Moreno, Borod, Welkowitz, & Alpert, 1990). However, Schwartz, Ahern, and Brown (1979) recorded bilateral corrugator and zygomatic EMG during a mood induction task. They found that subjects expressed positive emotions more intensely on the right side of the face and negative emotions on the left side of the face. However, the majority of research investigating emotional expressivity in normals and patients with focal lesions

supports the global rather than the bivalent model (Blonder, et al., 1991; Borod et al., 1985, 1988; Buck & Duffy, 1980).

Emotional arousal/activation

Hemispheric activation during emotional responding in normal subjects have been investigated using measures such as electroencephalography (EEG) and lateral eye movements (LEM). Using EEG, it has been found that in the frontal zones, positive emotions produced more left than right hemisphere EEG activation, while negative emotions produced more right than left EEG activation (Ahern & Schwartz, 1985; Tucker, Stenslie, Roth, & Shearer, 1981; Davidson et al., 1979; Davidson, et al., 1990). In addition, Ahern and Schwartz (1985) found that the right parietal zone was related to emotional intensity, whereas Bennett, Davidson and Saron (1980) as well as Davidson and colleagues (1990) found no differences in parietal activation related to emotion.

Lateral eye movements (LEM) have also been used as a measure of hemispheric activation. LEM towards the right have been interpreted as reflecting left hemisphere activation, while LEM to the left is suggestive of right hemisphere activation. Initial findings revealed more LEMs to the left during emotional experience (Davidson & Schwartz, 1976; Schwartz, Davidson, & Maer, 1975; Tucker, Roth, Arneson, & Buckingham, 1977). Ahern and Schwartz (1979) investigated lateral eye movement in response to

reflective questions in normal subjects. They found that positive emotional questions evoked more LEMs to the left. They interpreted this as left hemisphere specialization for positive emotions and right hemisphere specialization for negative emotions. However, the lateral eye movement methodology has been criticized (Erlichman & Weinberger, 1978).

Research on mood

Observation of mood after hemispheric damage has also been viewed as supporting the bivalent model. Sackheim et al. (1982) reported that pathological laughing was more likely to be associated with RHD and pathological crying was associated with LHD. Additionally, they found that patients with right hemispherectomies were judged to be euphoric in mood, while patients with left hemispherectomies were not. Also, they examined published case reports of gelastic epileptics, typified by laughing outbursts during ictal experience, with either left or right lateralized ictal foci. They found that ictal foci in gelastic epileptics was predominately left-sided. Based on previous literature, the authors suggested that the laughing outburst which occurred during ictal experience were caused by hyperactivity in the focal area. These authors concluded that both disinhibition and excitation cause different manifestations in mood in the right and left hemispheres.

Robinson and his colleagues have investigated depressive symptoms following stroke in both right and left hemisphere patients. In two studies, Robinson and Price (1982) and Robinson et al. (1984) found that patients with left hemisphere strokes were more depressed than patients with right hemisphere strokes. Starkstein, Robinson, and Price (1987) also noted that right hemisphere patients were indifferent and sometimes euphoric immediately following stroke. Additionally, Robinson and Szetela (1981) reported that patients with traumatic brain injury, while equally as impaired cognitively and physically, were not as depressed as stroke patients. Consequently, frequency and severity of depression is not solely related to amount of physical and cognitive impairment.

Differences in mood depending on caudality (anterior versus posterior location) of the lesions were also observed (Robinson et al., 1984). The left anterior group showed significantly more overall depression than the left posterior group, whereas the right posterior group were more depressed than right anterior group. Similarly, Starkstein et al. (1987) reported that when depression was present in RHD patients, it was associated with parietal lesions. Additionally, depression was found to be correlated with closeness of the lesion to the frontal pole (Robinson & Szetela, 1981; Starkstein, Robinson, and Price, 1987).

In a subsequent study, Sinyor, et al. (1986) assessed both cognitive and vegetative signs of depression using a variety of verbal report measures in unilateral stroke patients. Contrary to the above findings, no overall differences in depression were found between groups. However, consistent with the above findings, severity of depression in LHD patients was positively related to proximity of the lesion to the frontal pole. In addition, a curvilinear relationship was found for RHD patients such that both anterior and posterior lesions were associated with depression. Moreover, House et al. (1990) reported that RHD patients may be depressed more than is believed, but due to their deficits in emotional communication, their depression goes undetected.

Taken together, the results are equivocal. There is evidence in support of differential moods in left and right hemisphere damaged patients. Some investigators suggested that RHD patients express enhanced cheerfulness (e.g., Terzian, 1964), and LHD patients express or report experiencing more depression than RHD patients (e.g., Robinson et al., 1984). However, other investigators found no differences in depressed mood between LHD and RHD patients. Additionally, some studies revealed that during negative emotion, greater EEG activation was associated with anterior right activation. In contrast, during positive emotion, greater EEG activation was associated with anterior

left activation. However, EEG activation of right frontal and right parietal regions was associated with emotion intensity. Also, inferring hemispheric activation using LEM, findings supported greater right hemisphere activation during negative emotion experience and left hemisphere activation during positive emotion experience, but LEM methodology has also been criticized.

Specific bivalent models

In general, the bivalent model posits that the left hemisphere is specialized for positive/approach emotions and the right hemisphere is specialized for negative/avoidance emotions. However, there are many variations of the general bivalent model. Kinsbourne and Bemporad (1984) suggested that the left fronto-temporal cortex exerts action control, defined as manipulating external stimuli. They argued that left posterior parietal cortex sends exteroceptive input to the left fronto-temporal cortex. The right fronto-temporal cortex, on the other hand, controls emotional, internal arousal, while the right posterior cortex relays interoceptive information to the emotional control system. Consequently, in patients with right focal lesions, meaningfulness of environmental stimuli is deficient. Thus, RHD patients experience inappropriate emotionality. Additionally, Kinsbourne and Bemporad explained that the RH is specialized for monitoring both positive and negative emotional valence, but positive states enhance motivation

and readiness to act which are left hemisphere attributes. Specifically, passivity and involvement in perceptual judgement relates to RH activation, whereas overt responses or covert response planning is associated with left hemisphere activation.

Davidson and his colleagues (Fox and Davidson,1984; Davidson, 1985; Davidson et al., 1990) proposed a similar theory. They purported that the behavioral dimension of approach-withdrawal is the organizing dimension for hemispheric specialization in that the right hemisphere is specialized for withdrawal emotions such as disgust, whereas the left hemisphere is specialized for approach emotions such as interest. In addition, Davidson (1985) postulated there are reciprocal relations between the frontal and parietal lobes. Specifically, left frontal activation is balanced by right parietal activation and vice versa. For example, he stated that spatial cognition (right parietal) and positive affect (left frontal) are more likely to occur concurrently than verbal cognition (left parietal) and positive affect.

Heller (1990) posited a similar view. She asserted that the right hemisphere may be specialized for interpretation of emotion, but not specialized for the regulation of mood. Heller also emphasized the importance of distinguishing between the functions of the anterior and posterior regions of the brain, citing evidence that the

right temporal parietal regions are involved in interpretation of emotional information and evidence that implicates the frontal regions of both hemispheres in the experience of mood. Heller (1990) stated that the right parietal cortex mediates both cortical and autonomic arousal, while bilateral frontal regions mediate valence. She purported that experience of emotion is associated with patterns of activation in frontal and parietal brain regions.

Summary

As reviewed in the preceding sections, most evidence supportive of the bivalent model has been derived from two lines of research. These include: (a) findings of different mood reactions following right versus left hemisphere lesions, particularly those involving the anterior regions; and (b) findings in normals of hemispheric EEG activation asymmetries during induction of positive versus negative mood. In contrast, data from neuropsychological studies of affect perception are more in line with the view that the right hemisphere is critically involved in appraising nonverbal emotional signals, regardless of their valence. The discrepancy between such studies corresponds to the distinction raised by Heller (1990) between interpretation of emotion (viewed to be right hemisphere dependent) versus the regulation of mood (which is not viewed to be right hemisphere specific).

Observations that RHD patients are autonomically hypoaroused in response to affective scenes (relative to NHD and LHD patients) have been interpreted as support for a dominant role of the right hemisphere in emotional arousal. However, this interpretation is not without question given that such studies have generally measured autonomic responsivity only in response to neutral and unpleasant scenes (Meadows & Kaplan, 1992; Zoccolatti et al., 1982) or situations (Heilman et al., 1978). Pleasant scenes or stimulus materials have not been used in such studies and it remains unknown whether stroke of the right hemisphere equally attenuate autonomic reactivity to pleasant scenes. In and of itself, the current existing data that RHD stroke patients are hypoaroused to negative-affective scenes are equally consistent with the bivalent as well as the global right hemisphere model. Of relevance, Morris et al. (1991) recently reported valence-specific hypoarousal in a patient following a right temporal lobectomy. Skin conductance responses were obtained to unpleasant (mutilations), pleasant (attractive nudes), and neutral (breadbaskets) slides. This patient showed abnormally reduced SCR to unpleasant but normal SCR to pleasant and neutral slides, a pattern of findings that is consistent with a bivalent model. Had only unpleasant scenes been used in this study one would not be able to logically distinguish between the bivalent and global right hemisphere model. For this

reason, it is crucial to include both pleasant and unpleasant scenes or situations when studying psychophysiological responses in neuropsychological investigations of emotion. Such was employed in this study.

Before discussing the proposed study more fully, a brief overview of relevant literature on emotional psychophysiology will be presented. This is being done since the current study will include several psychophysiological indices (i.e., skin conductance, heart rate, facial electromyography) for assessing emotional responsivity in patients with right or left hemisphere lesions.

Emotional Psychophysiology

Autonomic Responding

At the psychophysiological level, the relationship between autonomic activity and emotion has been recognized for centuries. Recent technological advances have made the prospect of online physiological measurement more feasible. Theorists have attempted to understand the factors which influence skin conductance and heart rate. Sokolov (1963) described two types of responses which occur during conditioning: orienting and defensive reactions. He purported that the purpose of the orienting response (OR) is to increase sensitivity to incoming stimuli and that it includes both a transient increase in skin conductance. The defensive response (DR), on the other hand, is evoked in

response to high intensity or aversive stimuli and helps the organism to limit activity with the stimulus. This response includes increases in sympathetic activity such as cephalic vasoconstriction and increase in skin conductance.

Lacey and Lacey (1970) extended Sokolov's views of autonomic responding. They suggested that heart rate acceleration (tachycardia) during acute affective states is not a index of arousal per se, but reflects instead the organism's attempt to limit or terminate bodily turmoil produced by some stimulus. By contrast, heart rate deceleration (bradycardia) is induced with intention to respond to a task, attention to stimuli, and during vicariously experienced stress. Thus, Lacey and Lacey argued that the cardiovascular system is not a nonspecific index of arousal, but a highly specialized response mechanism which is integrated with affect and cognition and which also reveals individual differences in the way people deal with the environment.

Graham and Clifton (1966) pointed out that Sokolov (1963) and the Laceys (1958) agreed on the existence of an orienting and defensive response. However, Graham and Clifton indicated that they did not agree on the relationship between orienting and defensive responses and heart rate. Sokolov inferred that heart rate (HR) acceleration was related to increased sensitivity of incoming stimuli, whereas HR deceleration was related to

decreased sensitivity of incoming stimuli. The Laceys hypothesized the reverse pattern. In their thorough review of the literature, Graham and Clifton concluded that, in fact, the Laceys hypotheses have been supported in that HR deceleration is associated with orienting and HR acceleration is associated with defensive responding.

A large body of research exists in which the autonomic correlates of affective states have been investigated. Throughout the second half of this century, researchers have systematically explored the relationship between emotion and psychophysiological measures including skin conductance and heart rate. Early studies of systematic desensitization in phobic patients revealed that as the subjects imagined more fearful images, HR and skin conductance responses (SCR) increased (Lang, Melamad, & Hart, 1970).

In the late 1960s and early 1970s, a series of studies by Hare and colleagues indicated that slides of mutilated bodies evoked HR deceleration, an orienting response (OR). These results were initially confusing because it had been hypothesized that the slides would evoke fear and HR acceleration, a defense response (DR). Upon reanalyzing his data (Hare, 1972), it was found that some subjects had consistently reacted with HR acceleration, some with marked deceleration, and some with moderate deceleration.

Subsequently, researchers explored the differing reactions of phobics and nonphobics in response to affective

slides. The findings indicated that presentation of a feared object resulted in initial HR acceleration, e.g., (DR), while presentation of a nonfeared object results in HR deceleration, e.g., (OR) (Hare, 1973; Klorman, Weissberg, & Wiesenfeld, 1977; Klorman, Wiesenfeld & Austin, 1975). Additionally, SCR was elevated with the presentation of fearful stimuli (e.g., Klorman, Weissberg, & Wiesenfeld, 1977) and, in some studies, the amount of elevation was higher for phobics (Klorman, Wiesenfeld & Austin, 1975).

Imagery has also been used to evoke emotional states. It is important to note that during imagery, autonomic responsivity (i.e., HR and SCR) is influenced not only by the affective state, but also by other factors such as imagery instructions and the subjects' ability to image (Lang, Kozak, Miller, & Levin, 1980; Miller, Levin, Kozak, Cook, McLean, & Lang, 1987. Vrana, Cuthbert, and Lang (1986) found that normal subjects verbally reported experiencing more arousal, more unpleasantness, and less control during fear imagery than during neutral imagery. Fear images also evoked HR acceleration which lasted over a 10 second period. In contrast, neutral images produced acceleration followed by deceleration. Thus, HR and subjective report distinguished fearful from neutral imagery.

Taken together, the results of these studies are consistent with the views of Graham and Clifton (1966) and

Lacey and Lacey (1970). Heart rate typically increases in response to feared stimuli when presented visually or imagined. On the other hand, HR deceleration follows the visual presentation of a novel or interesting stimulus, whereas imaging of a novel or interesting stimuli produces HR acceleration followed by deceleration.

Facial Electromyography (EMG)

Before describing the facial electromyography research, the neuroanatomical pathways involved in facial muscle movements will be briefly reviewed. Motor neurons send information from the brain to innervate muscle and can be distinguished from sensory neurons which bring information to the brain. There are two types of motor neurons: upper motor neurons (UMN) and lower motor neurons (LMN). Upper motor neurons carry impulses from motor centers in the brain to the brain stem and spinal cord. Lower motor neurons carry information from brain stem and spinal cord to muscles. At the UMN level, fibers from either the contralateral or both hemispheres supply impulses to the LMN nucleus, the motor nucleus of the facial nerve, which innervates muscles of facial expression. The voluntary and involuntary motor pathways mediating facial expression are distinct from one another. Voluntary movement is mediated by the corticobulbar tract, originating in the precentral gyrus of the motor cortex of the frontal lobe. The involuntary pathway includes the basal ganglia, red nucleus,

and midbrain reticular formation. (Rinn, 1984). Although the pathways of voluntary and involuntary emotions are different, the measurement of facial expressions are the same regardless of the volitional quality of the expression.

Detailed facial coding systems, such as Ekman's FACS (Ekman & Friesen, 1978) and Izard's MAX (1978) have been used to measure minute muscle movements of the face. Because these rating systems are quite time intensive and because spontaneous facial muscle activity is often brief and too small to be observed overtly, facial electromyography (EMG) has sometimes been used to measure subtle changes in muscle movements. The most common facial muscle regions measured using EMG are the corrugator supercilli (brow) and zygomatic major (cheek) muscles regions. Various methods have been used to induce emotional states while EMG of the corrugator and zygomatic muscles have been measured. These emotion eliciting procedures have included imagery, viewing affective slides, self-referential statements, and self-disclosing interview. Consequently, the facial expressions that accompany these emotion induction procedures involve involuntary/spontaneous facial movements. The UMN innervation of the corrugator muscle is bilateral, whereas the UMN innervation of the zygomatic is contralateral (Rinn, 1984). Thus, muscle activity in the left and right corrugator regions cannot be activated

independently, but muscle activity of the left and right zygomatic regions can be stimulated separately.

During affective imagery, positive emotional states have been associated with decreased corrugator and increased zygomatic activity. Conversely, negative emotional states have been associated with increased corrugator activity and decreased zygomatic activity (Schwartz et al., 1976a, 1976b). Also, when verbal report of emotions has been obtained, corrugator activity positively correlates with unpleasant emotions and negatively correlates with pleasant emotions. The opposite pattern has been found for zygomatic activity (Brown & Schwartz, 1980; McCanne & Anderson, 1987; Slomine and Greene, 1993). Similar results have been reported from other investigators using self-referent statements designed to induce either elation or depression (Sirota, Schwartz, & Kristeller, 1987), and affective slides (Cacioppo, Petty, Lasch, and Kim, 1986). Additionally, an interview technique was employed to elicit and investigate naturally occurring emotional states (Cacioppo, Martzke, Petty and Tassinary, 1988). Replicating previous findings, elevations in corrugator EMG were related to lower positive emotion ratings and higher negative emotional ratings.

In sum, the above studies attest to the importance of the covert activity of the corrugator supercilli and zygomatic major muscles as indexes of emotion. Specifically, EMG activity of the corrugator supercilli has

been consistently found to increase during exposure to stimuli rated as unpleasant or during the reported experience of unpleasant affect. Conversely, activity of the zygomatic major has been found to increase during the report of positive emotional states.

Facial and Autonomic Studies

Few studies have included measures of both facial and autonomic responding. In one study of affective slides viewing, Greenwald, Cook, and Lang (1989) examined emotional ratings, HR, SCR, zygomatic and corrugator EMG. Zygomatic activity was positively related to pleasure ratings and corrugator activity was negatively related. Zygomatic EMG, however, also increased during unpleasant slides viewing. Neither muscle site was related to arousal ratings. Phasic HR acceleration was positively related to valence ratings, but not arousal. This relationship was weaker than the valence/EMG relationship. Skin conductance responses were significantly related to increased arousal ratings, but not valence ratings. Quite similar results were found when autonomic and facial responding were measured during imagery (York, 1991; Bradley, Lang, & Cuthbert (1991) in that HR acceleration and SCR were larger for pleasant and unpleasant compared to neutral imagery, and corrugator EMG was higher for the unpleasant compared to pleasant and neutral imagery.

Ekman and colleagues have found that giving subjects either muscle-by-muscle instructions to contract voluntary

sets of facial expression and asking subjects to relive a past emotional experience produced similar autonomic changes, i.e., increases in HR and SCR (Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, and Friesen, 1990). These authors concluded that there are biologically innate affect programs which, when activated, provide instructions to multiple response systems including skeletal muscles, facial muscles, and the autonomic nervous system.

Taken together, the above research suggests that zygomatic EMG increases with reported pleasantness, and somewhat with extreme unpleasantness. Corrugator EMG increases with reported unpleasantness. Skin conductance responses are positively related to reported experience of arousal, which can be induced through pleasant or unpleasant emotional states. Heart rate, however, is variable and depends on many factors such as reported affect, type of evoking stimuli, and individual differences in responding. However, during the presentation of emotional slides, HR acceleration is positively related to valence, but acceleration may be associated with aversive rather than pleasant stimuli when phobics are presented with their fear object. During imagery, HR typically accelerates during both pleasant and unpleasant scenes. Additionally, voluntary facial expressions produce changes in the autonomic nervous system consistent with other tasks used to induce emotional experience.

Anticipation of Affective Stimuli

Anticipation of affective stimuli has also been used to elicit emotion. Lang, Ohman, and Simons (1978) described the triphasic response of cardiac activity during a 4-8 second anticipation period. They reported that the onset of the preparatory period is characterized by a brief deceleration (D1). The initial deceleration is followed by an acceleratory peak (A1). Lastly, a deceleration occurs which lasts until the end of the preparatory interval (D2). D1 is observed when subjects are presented with single pure tones which are not followed by other stimuli and is thought to be an index of orientation. The acceleratory phase is seen in response to an abrupt stimulus or single stimulus with an uncomfortable intensity level. A1 has been interpreted as an index of a defensive reflex. It has also been evoked in the absence of noxious stimuli and during problem solving or mentation.

According to Lang et. al (1978), most investigators interpret the second deceleration, D2, as an index of anticipation of an overt response. D2, however, has been conditioned in classical conditioning paradigm even though no motor response is required. Consequently, D2 has also been viewed as an index of an attentive set. Similar HR patterns have been found by Simons, Ohman, and Lang (1979) in response to anticipation of slides (Simons, Ohman, & Lang, 1979; Klorman & Ryan, 1980).

There is a large body of literature based on anticipation of aversive stimuli. In one study, cluster analysis was used to identify different patterns of HR responses during anticipation of aversive noise (Hodes, Cook, & Lang, 1985). Results indicated that there were three types of responders; accelerators, decelerators, and moderate decelerators similar to the groups obtained by Hare (1972). The authors concluded that both the accelerators and decelerators developed the expectancy that the CS+ would precede the presentation of UCS. Accelerators, however, associated fear with the CS+, while the decelerators did not. The authors suggested that because the classical aversive conditioning paradigm specifies no overt response set, the subjects spontaneously assumed a response disposition. Specifically, some responded with an anticipatory, attentive set demonstrated by decelerators, whereas others displayed an implicit avoidance characterized by a defensive response. Moderate decelerators showed discordance between verbal and physiological behavior. Thus, these subjects maintained an attentive set, but evaluated the stimuli as aversive instead of interesting. The authors concluded that "It is conceivable that the tactile assault of shock is necessary to consistently elicit DR's to such potentially skin mordant stimuli as snakes and spiders," (p.555).

Psychophysiological responses of HR and skin conductance have been measured during anticipation of electric shock. Deane (1961) found that during anticipation of shock, HR accelerated over the baseline level. Additionally, in the groups who expected to receive shock when a 'target' number was presented, there was HR deceleration immediately preceding that number, even though, in one of these groups no shock had ever been received. These finding have been replicated (Elliot, 1966; Deane, 1969; Hodges & Spielberger, 1966). Threat of electric shock has also been found to produce increases in SCR (Bowers, 1971a, 1971b). Positron emission tomography (PET) measurements of regional blood flow have also been obtained during anticipation of electric shock (Reiman, Fusselman, Fox, & Raichle, 1989). Reiman and colleagues found that during anticipatory anxiety, there was significant blood flow increases to both temporal poles.

The investigation of the psychophysiology of pleasant and appetitive anticipation has received minimal attention in the experimental human literature. Consequently, psychophysiological responding during pleasant anticipation must be inferred from other studies. Based on the results of the above literature, it is likely that anticipation of pleasant stimuli would evoke physiological changes similar to those found during presentation of pleasant stimuli (i.e., increased zygomatic EMG and SCR). Also, based on the

above studies of anticipation during nonaversive anticipation (Simons, et al., 1979; Klorman & Ryan, 1980), HR is primarily deceleratory.

Summary

Psychophysiological measures of heart rate (HR), skin conductance responding (SCR), corrugator electromyography (CEMG), and zygomatic electromyography (ZEMG) have all been used as indices of emotional psychophysiology. Alone, each of these measures has been associated with various psychological phenomenon. For example, SCR has been associated with mental effort, attentive movements or attitudes, painful stimuli, variations in respiratory rate, along with emotional arousal and various other psychological phenomenon (Cacioppo & Tassinary, 1990). Increased heart rate has also been associated with various psychological phenomenon including startle, mental effort, and defensive responding (Cacioppo & Tassinary, 1990). In addition, corrugator electromyography has been associated with concentration as well as unpleasant emotional experience (Cacioppo, Petty, & Morris, 1985).

Because changes in heart rate, skin conductance, and facial EMG have all been found to be associated with psychological phenomenon other than emotional experience, changes in one of these variables is not necessarily indicative of emotional experience. However, examination of multiple variables over time has revealed specific

physiological response patterning which results in a one-to-one relationship with experience of emotional states. Thus, it is necessary to investigate patterns of physiological behavior over time in order to infer the presence of a psychological phenomenon based on physiological responding (Cacioppo & Tassinary, 1990).

Critical Issues

As reviewed earlier, there are two opposing views of how the cerebral hemispheres differ in their contributions to emotional processing. However, the precise role played by each hemisphere remains unclear. Some investigators have proposed that the right hemisphere is globally involved in all aspects of emotional processing including evaluation, expression, activation, and experience of emotion (Heilman et al., 1985). Others researchers have suggested that each hemisphere is specialized for a different type of emotion (Fox & Davidson, 1984; Kinsbourne & Bemporad, 1984; Tucker, 1981; Heller, 1990). The most popular version of the bivalent view is that the left hemisphere is dominant for positive/approach emotions, while the right hemisphere is dominant for negative/avoidance emotions.

Along with differences in laterality of emotional processing, investigators have speculated about differences in emotional processing based on caudality, i.e., anterior versus posterior regions of the brain. For example, studies of interpretation of emotional information implicate the

right temporal and parietal regions (e.g., Bowers et al., 1987), whereas studies of emotional mood have implicated the frontal lobes (e.g., Davidson, 1984).

Heller (1990) has interpreted the literature in terms of type of emotional processing, such that "cold" or nonexperienced emotional processing is modulated by the right posterior region. In addition, she posited that "warm" or experienced positive emotion is processed predominantly by the left hemisphere, whereas "warm or experienced negative emotion is processed predominantly by the right hemisphere. According to Heller, the majority of evidence in support of the right hemisphere model of emotion comes from studies which have investigated cognitive processing of information in brain damaged and normal subjects, whereas most evidence in support of the bivalent models of emotion has been derived from lateralization of mood states. Heller suggested that there is no reason to assume that because a hemisphere is associated with a particular mood state, that it must be specific for cognitive representations of that emotion.

In order to distinguish among the ability of the global and bivalent models to explain emotional experience, it is necessary to evoke emotion with both positive/approach and negative/withdrawal emotions. Because RHD patients have difficulty interpreting emotional stimuli, including faces and prosody (e.g., Bowers et al., 1987), it is difficult to

evoke emotional states in the laboratory. Thus, using an in vivo situation in which nonverbal emotional stimuli do not have to be interpreted would be useful in evoking emotion in RHD patients. Ideally, it is crucial for positive and negative emotions to be equally arousing. Unfortunately, it is difficult to equate in vivo positive and negative emotional experiences in emotional arousal because highly arousing negative emotional experience is much easier to experimentally induce than highly arousing positive emotional experience.

It is important to define emotional experience and how it can be measured. As mentioned above, emotional experience is defined as a phenomenon which can be indirectly measured using physiological measures (e.g., HR and SCR), overt behavior (e.g., facial expression, in this case measured using CEMG and ZEMG), and verbal report (e.g., paper and pencil assessment measures). In normal subjects these three response systems have usually been found to be concordant; however, discordant responses have been revealed in pathological populations (Patrick, Bradley, & Lang, 1991). These discordant results may imply that the three response systems are mediated by different subsystems. In brain damaged patients discordance is often observed. For example, patients with pseudobulbar laughter display overt behaviors of emotion, but verbally deny feelings associated with emotion (Heilman, Bowers, & Valenstein, 1993). These

results imply that there is a defect in the mediation output systems, such that behaviorally the patient responds, but without the corresponding subjective experience of emotion.

Due to the inability in directly measuring subjective experience, the ability to interpret discordance in response systems is weakened. To illustrate, two groups, A and B, are investigated during emotion-eliciting experiences. Both A and B verbally report experiencing emotion. However, group A does not exhibit psychophysiological measures indicative of emotion. Are the subjective emotional experiences of group A and B different? There are two possible interpretations: (1) they are experiencing qualitatively different emotional experiences, such that group A's experience of emotion is more "cognitive" than group B's experience, or (2) they are experiencing the same emotional experiences, but group A has a problem with the feedforward system of emotional psychophysiological responding. Because interpretation includes inferences about subjective experiences, neither interpretation can be proven correct or rejected as invalid. It is unclear, at this time, how patients with unilateral damage experience emotion based on the interaction of these three response systems. Specifically, it is unknown whether unilateral lesions would produce concordance or discordance of emotional experience.

It is important to consider the constraints that are placed on evaluating emotional experience in patients with focal lesions. For example, left hemisphere damaged patients often have difficulty with language, which may affect their verbal report data. To minimize this problem in the present study, severely aphasic patients would not be used and only verbal report measures with simple language were used. Also, right hemisphere damaged patients often have difficulties with visual attention, neglect, and vigilance. Consequently, adequate attention to the task at hand must be insured among RHD patients.

To study emotional experience, it is important to measure all three response systems; verbal report, overt behaviors, and physiological indices. One way to better understand the neuropsychology of emotional experience is to use paradigms which are highly sensitive to emotional responding. The present study focused on an anticipation paradigm (Reiman et al., 1989) designed to investigate verbal report, heart rate, skin conductance, and facial responses associated with emotion. In order to examine the psychophysiology of emotional experience, an "in vivo" situation was used. Using anticipation of "in vivo" aversive and pleasant stimuli, it was easier for patients to interpret the emotional meaning of the situations because they did not have to analyze the affective quality of various perceptual stimuli.

CHAPTER 2
STATEMENT OF THE PROBLEM

The purpose of the present study was to broadly examine emotional responsivity of RHD and LHD patients in affect-evoking situations and determine whether the pattern of responses obtained from these patients was more in line with predictions of a global right hemisphere model versus a bivalent hemisphere emotion model. To examine this verbal report, autonomic measures of arousal (SCR, HR), and indices of facial muscle movement (EMG) were be collected in situations that are known to elicit negative (anticipation of shock) and positive responses (anticipation of reward) in normals.

To date, few neuropsychological studies of emotion with focal lesion patients have concurrently investigated more than one component of emotional responsivity. That is, either autonomic indices have been obtained (Heilman, Schwartz, & Watson, 1978) or verbal report of mood states have been obtained (Robinson & Price, 1982). No study to date has used facial EMG to examine emotional responsivity in focal lesion patients. Facial EMG may potentially be a useful tool in that it has been shown to be sensitive to

changes in the reported experience of valence in normal individuals (Greenwald et al., 1989).

Further, those patient studies that have examined psychophysiological indices of arousal in response to emotional stimuli have typically used perceptual stimuli (i.e., affective scenes) which must be accurately "interpreted" in order to induce emotion. Patients with RHD are known to have an array of visuoperceptual and hemispatial attentional scanning difficulties which can potentially interfere with their interpretation of such stimuli. Consequently, findings that RHD patients are autonomically hypoaroused in response to emotional scenes may, in part, be secondary to difficulties in interpreting these stimuli.

To avoid such confounding, the present study used "in vivo" situations to elicit negative and positive emotions among focal lesion patients. An anticipatory anxiety paradigm adopted from Reiman et al. (1989) was used to induce negative emotion (i.e., anxiety). In this paradigm, subjects are told that they would sometimes receive a mild shock. Findings with normals reveal changes in autonomic arousal during the period that the subject is awaiting shock in conjunction with self reports of increased levels of anxiety (as measured by the State-Trait Anxiety Inventory). An anticipatory reward paradigm was used to induce positive emotion. Here, subjects were told that they would sometimes

receive monetary reward (i.e., dollar bills or lottery tickets).

The specific objectives of this study are to determine: (a) whether patients with RHD or LHD become autonomically aroused in these in vivo emotional situations (as indexed by HR and SCR changes); (b) whether they display contraction of facial muscles (as measured by EMG indices) that correspond to the positive-negative nature of the emotional situation; and (c) whether they explicitly report subjective changes in their emotional experiences (as measured by their responses to questionnaires).

According to the global right hemisphere emotion model, the RHD patients should display attenuated responsivity across all three response domains (arousal, facial, verbal report) in both the negative and positive emotion-eliciting situations. In other words, relative to the LHD group, the RHD patients should be less autonomically aroused, show minimal facial muscle contractions, and report less intense changes in their subjective experience of emotion. Diminished responding by RHD patients would be observed in both the anticipatory anxiety paradigm, as well as the anticipatory reward task.

According to the bivalent hemisphere emotion model, the responses of the RHD and LHD patients would vary as a function of the positive-negative nature of the induced emotional situation. Specifically, the RHD group would show

diminished autonomic responsivity and less intense reports of emotional experience in the anticipatory anxiety task relative to the anticipatory reward task, whereas the LHD group would show the opposite pattern.

Overview of Experimental Design

Patients with RHD, LHD, and NHD participated in two experiments. Both experiments consisted of two parts, an anticipatory anxiety task and an anticipatory reward task. In the first experiment, a two-stimulus paradigm (see Vrana, Cuthbert, & Lang, 1989) was used in both the anticipatory anxiety and anticipatory reward tasks. Specifically, one warning tone signaled that the subject would receive shock stimulation during the subsequent six seconds, whereas the other tone signaled that shock would not occur. Prior to the beginning of the task, subjects learned which tone would be associated with shock and which with no shock. An analogous two-stimulus paradigm was used in the anticipatory reward task. Psychophysiological measures of arousal (HR, SCR) and facial EMG (corrugator and zygomatic) were obtained during the six second anticipatory interval.

In the second experiment, a slightly different paradigm was used to examine anticipatory anxiety and reward in RHD and LHD patients. Specifically, there was a 5 minute interval (versus 6 seconds in Experiment 1) during which the subject awaited shock (or reward). Five-minute control trials were also be given in which the subject is told that

no shock (or reward) would be presented. During these 5-minute anticipatory intervals, subjects were administered brief mood questionnaires (i.e., Positive and Negative Affect Schedule and Self-Assessment Manikin).

The use of the 5-minute paradigm in Experiment 2 is more suitable for obtaining self-report information, whereas the use of 6-second two-stimulus paradigm in Experiment 1 is more suitable for obtaining reliable brief psychophysiological indices of emotion.

Hypotheses and Predictions

Overall Hypotheses

According to the global right hemisphere model, emotion is modulated predominantly by the right hemisphere. Consequently, the global model hypothesizes that patients with RHD will display attenuated responsivity, relative to the LHD group, across all three response domains (arousal, facial, and verbal report) in both negative and positive emotion-evoking situations.

In contrast, the bivalent model posits that positive/approach emotions are mediated by the left hemisphere and negative/avoidance emotions are mediated by the right hemisphere. According to the bivalent model, the responses of the RHD and LHD patients would vary as a function of valence (positive-negative nature) of the induced emotion. Specifically, the RHD group would show diminished responses in all three response domains (arousal,

facial, and verbal report) during the anticipatory anxiety (negative emotion) situation relative to their responses during anticipatory reward (positive emotion). The LHD group would show the opposite pattern.

Specific Predictions for Experiment 1: Psychophysiological Arousal and Facial EMG during Anticipatory Anxiety and Anticipatory Reward in Patients with RHD and LHD

Normal control group (NHD)

In line with previous research, it is anticipated that the normal control group (NHD) will experience unpleasant emotion (anticipatory anxiety) during the shock anticipation condition and more pleasant emotion (anticipatory reward) during the prize anticipation. Specific predictions regarding psychophysiological responsivity (HR, SCR) and facial EMG are derived from empirical research with emotion-inducing stimuli. A replication of previous findings is expected such that:

1. Compared to baseline HR, a HR triphasic response (D1, A1, D2) will be observed during shock anticipation and prize anticipation. The A1, acceleratory peak, is expected to be greater during shock than during prize anticipation. In some subjects, however, deceleration only may be observed during prize anticipation. Relative to the experimental trials, attenuated HR change will occur during control trials.
2. Compared to baseline SCR, SCR will be greater during shock and prize anticipation compared to no shock/no

reward control trials. Additionally, SCR will decrease over trials.

3. Compared to baseline corrugator EMG, corrugator EMG (CEMG) will be elevated during shock anticipation and will remain relatively unchanged during prize and control trials.

4. Compared to baseline zygomatic EMG, zygomatic EMG (ZEMG) will increase during prize anticipation. Additionally, a smaller increase may be revealed during shock anticipation. Also, ZEMG will not change from baseline during control trials.

Focal Lesion Patients (RHD and LHD)

Predictions for the RHD and LHD patients differ depending on the global right hemisphere emotion model versus the bivalent model. Specific predictions for the right hemisphere emotion model will be first presented and then followed by those from the bivalent model.

A) Global Right Hemisphere Emotion Model: According to this view, patients with right hemisphere damage are relatively blunted in their emotional responsivity and experience of emotion. Thus, RHD patients will experience less anxiety and positive feelings during the shock and prize conditions, respectively, relative to the NHD and LHD subjects. In contrast, LHD patients may experience more intense emotional responsivity than NHD subjects. Specific predictions are as follows:

1. During shock and prize anticipation, LHD subjects will display similar or accentuated HR response patterns compared to normal controls, whereas RHD subjects will display decreased HR responding relative to normal controls. HR responding will be greater for the LHD and NHD groups during shock and prize trials compared to control trials. HR responding for the RHD group will not differ between shock, prize and no shock/no reward control trials.

2. During both shock and prize anticipation, LHD patients will display greater SCR than the normal controls. For the RHD patients, SCR will be smaller than that of the LHD and NHD patients. SCR will be greater during shock and prize trials than control trials for the LHD and NHD groups, whereas the difference in SCR for the RHD group between shock, prize, no shock/no reward control trials will be smaller.

3. During shock anticipation, corrugator EMG reactivity will be similar or greater for LHD compared to the NHD patients, whereas RHD patients will show smaller corrugator EMG compared to NHD patients. For LHD and NHD patients corrugator EMG will be greater for shock trials than no shock trials. However, differences in corrugator EMG will be smaller between shock and no shock control trials in RHD patients.

4. During prize anticipation, zygomatic EMG reactivity will be similar or greater for LHD compared to NHD patients, whereas RHD patients will show smaller zygomatic EMG compared to NHD patients. For LHD and NHD groups, zygomatic EMG will be greater for prize compared to no reward trials. However, differences in zygomatic EMG will be attenuated between prize and no prize control trials in RHD patients.

B) Bivalent Emotion Model: According to this view, patients with RHD should demonstrate attenuated anxiety during the shock anticipation condition (relative to NHD controls), and either normal or enhanced pleasant feelings during anticipatory reward condition. In contrast, patients with LHD should demonstrate attenuated pleasant feelings during the anticipatory reward condition (relative to NHD subjects) and either normal or enhanced negative feelings during the anticipatory shock task. These results may be most pronounced in patients with anterior-extending lesions. Specific predictions are as follows:

1. During shock anticipation, the LHD subjects will have greater or similar HR responding compared to the NHD group, whereas RHD subjects will have smaller HR responding relative to NHD subjects. Additionally, LHD and NHD patients will display greater HR responding during shock relative to no shock trials, whereas HR responding in RHD patients will not differ between

shock and no shock trials. During prize anticipation, RHD subjects will have greater or similar HR responding compared to normal controls, whereas LHD patients will have smaller HR responding relative to NHD patients. Also, RHD and NHD groups will display greater HR responding during prize relative to no reward control trials, whereas LHD patients will not differ between prize and no prize trials.

2. During shock anticipation, the LHD patients will have greater or similar SCR compared to NHD controls and RHD patients will have smaller SCR compared to NHD controls. Also, LHD and NHD subjects will have greater SCR during shock compared to no shock trials, whereas SCR will not differ between shock and no shock trials in RHD patients. During prize anticipation, however, RHD patients will have greater SCR than NHD patients, while the LHD patients will have smaller SCR than NHD subjects. Similarly, RHD and NHD patients will have greater SCR during prize compared to no prize trials, whereas SCR will not differ between prize and no reward trials in LHD patients.

3. During shock anticipation, the RHD subjects will have smaller CEMG compared to NHD patients, while the LHD group will have greater or equal corrugator EMG compared to NHD patients. Compared to control trials, LHD and NHD groups will show accentuated corrugator EMG

during shock trials, whereas RHD patients will exhibit no differences.

4. During prize anticipation, the RHD will have greater or equal ZEMG compared to the NHD group which will have greater zygomatic EMG compared to LHD group. Relative to no reward control trials, RHD and NHD subjects will display increased zygomatic EMG during reward trials, whereas LHD patients will show no differences.

Specific Predictions for Experiment 2: Subjective Report of Emotion during Anticipatory Anxiety and Reward Tasks by RHD, LHD, and NHD Patients

The hypotheses and predictions for this experiment are similar in kind to those of Experiment 1.

Normal control group (NHD)

1. In line with previous research, it is expected that the NHD group will report greater state anxiety during the shock than no shock control trials.
2. Similarly, during prize anticipation, NHD group will report more intense positive emotions than during the no prize control trials

Focal lesion patients (RHD and LHD)

A) Global Right Hemisphere Emotion Model: The predictions of this model are as follows:

1. During shock anticipation, the LHD and NHD groups will report greater anxiety (based on state anxiety scores on the State-Trait Anxiety Inventory, dimensional

ratings of unpleasantness, arousal, powerlessness on the Self Assessment Manikin, and the negative affect factor score of the Positive and Negative Affect Schedule) than the RHD group. The LHD and NHD groups will report greater state anxiety during shock than no shock control trials. The difference in reported anxiety will be attenuated in RHD patients between shock and no shock control trials.

2. During prize anticipation, the LHD and NHD subjects will report greater positive emotions (based on dimensional ratings of pleasantness, arousal, and dominance on the Self Assessment Manikin, and the positive affect factor score of the Positive and Negative Affect Schedule) compared to the RHD. LHD and NHD groups will report more positive emotions during prize compared to no reward trials. The difference in reported positive emotions will be smaller during prize compared to no reward trials in RHD patients.

B) Bivalent Emotion Model: Predictions based on the bivalent view are:

1. During shock anticipation, the LHD subjects will report greater or equal anxiety compared to the NHD patients, whereas RHD subjects will report less anxiety than the NHD group. More anxiety will be reported during shock compared to no shock trials for LHD and NHD patients.

RHD will report no differences in anxiety between shock and no shock trials.

2. During prize anticipation, the RHD will report more or equal positive emotion compared to the NHD patients, whereas LHD subjects will report less more positive emotions than the NHD group. Also, RHD and NHD subjects will report more positive emotion during prize compared to no reward control trials. LHD patients will report no differences in positive affect between prize and no reward trials.

CHAPTER 3
METHODS

Subjects

A total of 48 right handed patients were included in the study. Handedness was determined by Briggs and Nebes (1975) abbreviated version of Annett's (1970) questionnaire. The stroke patients were recruited through clinics, laboratories, and medical records at Shands Teaching Hospital at the University of Florida and the Veteran's Administration Hospital in Gainesville. Additionally, other subjects were recruited through neurologists, physical therapists, and stroke clubs in the north central Florida region. Control subjects were recruited through laboratories at Shands Hospital and the VA, volunteer services at the VA hospital, as well as from other local senior groups.

All subjects were alert, cooperative, and oriented to time, place, and person. The population consisted of four groups; 12 patients with right hemisphere ischemic infarctions (RHD), 12 patients with left hemisphere ischemic infarctions (LHD), and 24 patients without neurologic disease (12 were controls for the RHD group and 12 were controls for the LHD group). Attempts were made to match sex, age, and level of education across groups. There were

12 males in both the RHD and the RH NC groups. In the LHD and LH NC groups there were 11 males and 1 female within each group. Separate analyses of variance (ANOVA) were used with group (LHD, LH NCS, RHD, RH NCS) as the between subject factor to determine if there were any group differences in age and education. There was no significant difference in the age of the subjects between each group. The means and standard deviations for age of each group are as follows: RHD=63.01(9.74), RH NCS=63.92(10.63), LHD=66.75(7.59), LH NCS=68.67(7.35).

There was also no significant differences between the number of years of education for subjects between each group. The means and standard deviations of years of education for each group are as follows: RHD=13.08(3.97), RH NCS=14.25(2.83), LHD=12.79(2.60), LH NCS=13.83(3.95).

The ANOVA tables for the analyses examining age and education are presented below.

AGE

	SS	DF	MS	F	SIG of F
GROUP	238.729	3	79.576	.996	.404
ERROR	3514.750	44	79.881		

EDUCATION

	SS	DF	MS	F	SIG of F
GROUP	16.182	3	5.394	0.468	0.706
ERROR	507.563	44	11.536		

Any patient with a pacemaker was excluded. All subjects were questioned about hearing and visual defects. All medications taken by the subjects on the day of the psychophysiological measurements were recorded and a list of these medications is provided in Table B-1, B-2, B-3, and B-4 of Appendix B.

All subjects were administered the Zung Depression Rating Scale. No group differences were found in their self report of depression on the Zung [$F(3,41) = 2.134$, $P = .1107$]. The mean scores and standard deviations on the Zung are as follows: LHD (mean=38.636, sd=5.29); LH NCS (mean=36.091, sd=5.28); RHD (mean=40.167, sd=7.814); RH NCS (mean=34.091, sd=5.991).

The RHD and LHD subjects all had a CT or MRI performed for clinical purposes. To be included, patients had a discrete abnormal area compatible with cerebral infarction on the head scan. Patients with tumors, hemorrhages, trauma, or bilateral cerebral infarcts were excluded. All subjects were tested at least 5 months post stroke in order to control for possible changes in autonomic responsivity over time. A t-test was conducted to examine group differences in the amount of time since the last cortical stroke. No differences were found between the groups [$T(1,22) = .588$, $P = .5626$]. The average time in months for the LHD group was 78, sd=72.72 and the average time in months for the RHD group was 60.92, sd=69.59.

Using the atlas of Damasio and Damasio (1989), lesions from the patients' CT scans were projected onto templates by a board certified neurologist (K.H.), who was unaware of patients' performance. Based on their scans, the neurologist divided the stroke patients into anterior, posterior, and mixed groups. Lesions were termed "posterior" if located behind the central fissure or within the posterior temporal lobe. Lesions located in front of the central sulcus or involving the anterior temporal lobe were considered "anterior." Lesions were considered "primarily anterior" if they also involved the primary sensory areas or Heschl's gyrus and "primarily posterior" if they involved the primary motor areas. Lesions involving both anterior and posterior regions, and/or regions between them were considered "mixed."

All of the scans were then ranked from largest to smallest lesion by the neurologist. The rankings were analyzed using an independent samples Wilcoxon Test of Ranked Sums to explore the group differences in size of lesion. No significant differences was found between the RHD and LHD groups [$W = 141.0$, $P = 0.6075$].

A summary of the neurological information for each subject is provided in separate tables for each group. See Tables B-5 and B-6 in Appendix B.

Baseline Evaluation

The baseline evaluation included a review of neurological records along with a neuropsychological and psychophysiological screening. All patients' neurological records were reviewed by a neurologist prior to acceptance into the study. All patients psychophysiological responses to a series of 60db tones was assessed. The psychophysiological screening is described more fully in the procedure section for experiment 1. The neuropsychological screening is described below.

All patients were administered the Information and Similarities subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R), Wechsler Memory Scale-Revised (Orientation, Digit Span, Logical Stories I,II and Visual Reproductions I, II subtests), Benton Facial Recognition Test, Western Aphasia Battery (Spontaneous Speech, Auditory Comprehension, Repetition, and Naming subtests), Florida Neglect Battery (shortened version including line bisection, cancellation, visual extinction, tactile extinction, and draw/copy a clock). The average performance on these measures by group is presented in Table B-7. Individual subjects' performance on these measures are presented in Tables B-8, B-9, B-10, and B-11 in Appendix B.

T-tests were conducted to examine group differences in neuropsychological functioning. Examination of the WAIS-R subtests revealed that the LHD subjects had signicantly

lower scores on information compared with the CONS, but not the RHD subjects. There were no significant group differences on the similarities subtest of the WAIS-R. Both the LHD and RHD subjects had significantly decreased digit span forward and backwards compared to the CONs.

Results of memory testing revealed that the LHD subjects scores on immediate recall on Logical Memory were significantly lower than controls. However, after a delay, the RHD subjects had significantly poorer recall compared to the CONs. On both Logical Memory I and II, there were no differences between the LHD and RHD subjects. RHD subjects performed worse on Visual Reproductions I and II compared with CONs, but not LHD subjects.

Language testing revealed that the LHD subjects had more difficulty with comprehension and had a lower overall Aphasia Quiotent compared with CONs and RHD subjects.

All Ss were also administered the Florida Affect Battery. Their results on this test are provided in Tables B-12, B-13, B-14, and B-15 in Appendix B.

Experiment 1: Psychophysiological Arousal and Facial EMG during Anticipatory Anxiety and Anticipatory Reward in Patients with RHD and LHD

This experiment consisted of two parts, an anticipatory anxiety and an anticipatory reward task. In both, a two stimulus paradigm was used whereby subjects were told that one target tone would signal the occurrence of shock or reward during the following 6 seconds, whereas a second

target tone indicated that nothing would occur during the 6 second interval. Autonomic measures of arousal (HR, SCR) and facial EMG measures were obtained. The order of the anticipatory anxiety task and the anticipatory reward tasks was counterbalanced across subjects in each group.

Stimuli and Apparatus

The electrical stimuli was delivered by a Grass S88 Stimulator and Isolation Unit. A Zenith Data Systems AT clone computer was programmed to deliver one high tone (usually 800 or 1000 Hz) as a warning stimulus at 60 db for one second. The computer also interacted with the stimulator such that six seconds after presentation of a specific tone, a shock was administered. The presentation of a low tone (usually 400 or 600 HZ) was not followed by a shock. For the reward task, the computer produced one high and one low tone. Six seconds after the high tone, the screen produced a message stating how many dollars or lottery tickets the subject had won so far and a picture of a smiling face. Six seconds after the low tone, nothing occurred.

Stimulus presentation and data storage was controlled by customized application software. Equipment for recording heartbeat (HR), skin conductance rate (SCR), corrugator electromyography (CEMG), and zygomatic electromyography (ZEMG) included a set of Colbourn Instruments data

acquisition modules, a DT2805 Multifunction Board, and a Zenith Data Systems AT clone computer.

Heartbeat was monitored by a Colbourn Instruments EKG Coupler recorded from standard lead II. Colbourn Instruments Bipolar comparator was used to detect the R-peak of the EKG. Sampling occurred at 200 Hz. The output of the Schmitt trigger was sampled at the digital input port of a DT2805 Multifunction Board installed in a Zenith Data Systems AT clone computer.

Skin conductance was measured by attaching 4-mm Ag/AgCl electrodes to the thenar and hypothenar eminences of the palm ipsilateral to the lesion. To control for possible hand effects NHD subjects were divided into left hemisphere normal control (LH NC) and right hemisphere normal control (RH NC) groups. The LH NC group had electrodes placed on their left hand and the RH NCs had electrodes placed on their right hand. One LHD subject had skin conductance measured on his right hand because his left arm had been amputated. Since recent evidence (Tranel & Damasio, 1994) suggests that brain damage subjects do not display differential skin conductance between their right and left hands, it was decided to include this subject in the SCR analyses. A 0.05 m NaCl electrolyte (Johnson & Johnson K Y Jelly) was used. Colbourn Instruments Skin Conductance module S71-22 was used to condition the SC signal. This is a constant voltage system which passes 0.5v across the palm

during the recording. Sampling occurred at 20 Hz. The analog SC signal was then be digitized by the Multifunction board, which physically resides in the backplane of the Compaq computer. Software control was accomplished by customized programs.

Corrugator and zygomatic EMG was recorded using 2-mm Ag/AgCl electrodes placed unilaterally over the corrugator and bilaterally over the zygomatic muscle regions after the skin was cleansed with 70% EtOH. Zygomatic EMG was collected bilaterally because motoneuron pathways which innervate the lower face are largely contralateral (Rinn, 1984). On the other hand, corrugator EMG was collected ipsilaterally because motorneurons innervating the upper face muscles are for the most part, bilateral (Rinn, 1984). Additionally, to control for possible laterality effects, the LH NCs had electrodes placed over their left brow and the RH NCs had electrodes placed over their left. Muscle regions were designated using the placement specified by Tassinary, Cacioppo, & Geen (1989). Four Colbourn model S75-01 High Gain Bioamplifiers with bandpass filters were used to record the signals. Filter level was set at 90-1000 Hz and coupling at 10 Hz (Fridlund & Cacioppo, 1986). Data was integrated with Colbourn model S76-01 Contour Following Integrator with a time constant set at 500 milliseconds. Sampling rate was 20 Hz.

Procedure

At the beginning of each test session, there was approximately a 5 minute adaption period during which the recording electrodes had been applied and the subject relaxed while sitting in a comfortable chair in a climate controlled shielded room. Following this adaption period, basic physiologic reactivity (HR, SCR) to a series of 24 tones, in 8 blocks of three with two tones at 400 Hz and one at 100 Hz (each at 60 db for .5 seconds) was measured and the course of orienting and habituation was assessed.

The anticipatory anxiety paradigm adopted from Reiman et al. (1989) to induce negative emotion and reward portion of the study to evoke positive emotion were given independently and the order in which they were given was counterbalanced by subject for each group. For both the anticipatory anxiety and anticipatory reward, there was 40 trials: 20 control and 20 experimental shock or reward trials. Each trial began with a meditation period of 2 to 3 seconds, where subjects repeated the number one silently to themselves, followed by one of four tones (between 500 and 1500 Hz for 1 second at 60db). Physiological measurements were recorded during the last second of each baseline period through the six second interstimulus interval and through the six second stimulus and recovery period.

Anticipatory shock task

Before beginning the anticipatory anxiety task, each subject choose the intensity of shock. This was done by increasing voltage from 0 volts in five volt increments. Half second shocks were administered after each increase in voltage until the subject found the shock intensity "uncomfortable but not painful."

Before the onset of the session, subjects were told which of two tones corresponded to shock trials and which corresponded to control trials. This two-stimulus paradigm is similar to that used by Vrana, et al. (1989) in which a warning signal is followed six seconds later by an electric shock. The subjects were instructed that during the shock trials at tone offset (after hearing the high tone), there will be a 6 second interstimulus interval which will be followed by a shock. In addition, the subjects were told that when they heard the low tone, it signaled that in six seconds, nothing would happen.

Anticipatory reward task

At the beginning of the sessions, subjects were told which tone would indicate that they would receive a dollar (or lottery ticket) and which tone was not associated with reward. The higher tone always designated reward. The designated tone for the reward trials was followed by a six-second interval after which a message appeared on the computer screen. The message read "You have won -- dollars"

and a smiling face. The number on the message corresponded to the total number of dollars and/or lottery tickets won. As in the anticipatory anxiety task, the tone designating the control trials, the low tone, was not followed by anything.

For both the shock and reward tasks, a square appeared on the screen, during the 6 second period between tone and stimulus. A cross gradually enlarged within the square. By the end of the six seconds, the cross would touch each side of the square and the screen would go blank. Since it is unclear how patients with cortical strokes estimate time, the square and growing cross were used to control for time estimation by helping all of the subjects keep tract of time during the six second period.

During both the anticipatory shock and anticipatory reward tasks, the procedure was interrupted after each block of 10 trials. At that time, the experimenter entered the room and administered to the subjects the three-item Self-Assessment Manikin (SAM) (Hodes, Cook, & Lang, 1985). The SAM, which is described below, is designed as a self-report measure of valence (pleasantness-unpleasantness), arousal, and dominance (control).

The Self-Assessment Manikin (SAM) measures subjective ratings of three independent affective dimensions which have been derived from factor analytic studies (Hodes, Cook, & Lang, 1985). The three dimensions include valence (pleasant

to unpleasant), arousal (aroused to calm), and control (dominance to submission). There are both computer and paper and pencil versions of SAM. In this study, a paper and pencil version of SAM in which each dimension was presented as a series of five cartoon characters was be used. For the valence dimension, SAM's facial expression gradually changes from a smile to a frown. Arousal is denoted by increased activity in the abdomen to no activity and wide eyes to closed eyes. Control is represented from a very large character who gradually shrinks in size to a very small character.

During both the anticipatory shock and the anticipatory reward tasks, subjects were asked to rate how they felt using the SAM. This was done after each block of 10 trials, so that two ratings were obtained after each of the following conditions: shock anticipation, anticipation of no shock, reward anticipation, anticipation of no reward.

Data Reduction

Heart rate

First raw data was examined. Based on the subjects data as a whole, missing beats and double beats were estimated and corrected. Next, a computer program was used to more thoroughly determine missed beats and double beats. Double beats were removed from the data set. The computer used the surrounding beats to estimate the missing beats. Average half second beats/minute were obtained for each

condition for four blocks, each containing five control trials and five stimulus trials. An average baseline score was derived for the high and low tones for each trial block. Beats per minute change was then determined by subtracting the baseline value from each half second beats/minute average for each trial block. Those values were then used to designate average D1, A1, and D2 for each subject for the stimuli and control blocks within each condition. D1 was designated as the lowest point within the first 3 seconds. The highest point following D1 was considered A1. D2 was the lowest point following A1. If the last value in the six second period was the A1, D2 and A1 were the same.

Skin conductance

A computer program calculated baseline, skin conductance response (change from baseline), range-corrected skin conductance response scores (minimum and maximum values within each experimental condition was used in the calculations), and half recovery time. Data was divided into four blocks, each containing five control trials and five stimulus trials. One average range-corrected SCR was calculated for each stimuli and control block within each condition. Additionally, range corrected SCR was also recoded by changing all values under .02 micro ohms to zero. An average recoded range corrected SCR was calculated for each stimulus and control block within each condition.

Facial electromyography

A computer program calculated baseline corrugator electromyography (CEMG), left zygomatic electromyography (ZGL), and right zygomatic electromyography (ZGR) along with average CEMG, ZGL, and ZGR over the six second period for each block within each experimental condition. As a consequence, for each trial block, there was one baseline and one average score for each stimulus and control trial for each of the three facial muscles regions: CEMG, ZGL, ZGR. Difference scores for each of the variables was obtained for each block by subtracting the average score from the average baseline score for each subject.

Experiment 2: Subjective Report of Emotion During Anticipatory Shock and Reward Tasks by RHD, LHD, and NHD Patients

This experiment also consisted of two parts, an anticipatory anxiety task and an anticipatory reward task. Both were similar in kind to those of Experiment 1 except that a 5 minute anticipatory interval was used in this study in order to give the subjects time to complete verbal report questionnaires about their affective state during anticipation. In this experiment, the anticipatory shock task and the anticipatory reward task were counterbalanced by subject within each group.

Stimuli and Apparatus

The stimuli and apparatus used to dispense the shocks were identical to used in Experiment 1. Additionally, two

verbal report measures of affective states were given. These included the Self Assessment Manikin and the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988). The SAM was described in the methods for Experiment 1 above. The Positive and negative affect schedule is comprised of two 10-item mood scales. Using factor analysis positive affect (PA) and negative affect (NA) factors have been identified. The directions used were, "How _____ are you feeling right now?" The experiment inserted each item into the blank. Subjects were asked to rate the intensity of each feeling on a scale of 1 to 5, with 1 corresponding to "not at all" and 5 corresponding to "extremely."

Procedure

This study consists of two parts, an anticipatory shock task and an anticipatory reward tasks condition which were counterbalanced, and described below.

Anticipatory shock task

This task had two parts, a shock and a no-shock condition. In the shock condition, the subject waited five minutes to receive a shock. Subjects were told that they would receive a shock five minutes after hearing the warning tone and that the strength of this shock was either the same or greater than that previously given in Experiment 1. At the end of the five minutes, subjects were given the same intensity of shock they had previous received in Experiment

1. During the five minute anticipation period, negative emotions were assessed using the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988), and the Self-Assessment Manikin (SAM) (Hodes, Cook, & Lang, 1985). The experimenter read each item to the subjects and recorded the responses.

In the no-shock task, subjects waited for a five minute period with the understanding that they would not receive a shock. The no-shock control condition consisted of a five minute period during which time the subjects were administered the PANAS and SAM.

Anticipatory reward task

The reward condition consisted of counterbalanced reward and no-reward conditions. In the reward condition, subjects waited to receive a reward. During the reward condition, subjects were informed that they would receive between 5 and 8 dollars, lottery tickets, or a combination of both. Subjects were administered the PANAS and SAM while waiting for the reward. In the no-reward condition, subjects were informed that they were not receiving a reward. The same questionnaires were administered during the five minute no-reward condition.

Design Issues

A few problems inherent in the design of this project are presented here. First, it is presumed that the positive and negative emotions experienced in the anticipatory prize

and anticipatory shock situations will not be equal in intensity even for the NHD group. Specifically, intensity of anxiety/negative affect in anticipation of electric shock will probably be greater than the intensity of joy/positive affect in anticipation of a dollar or a lottery ticket. However, due to financial constraints, it is not possible to raise the financial value of the reward. Yet, by giving each subject the choice between a dollar and a lottery ticket, hopefully the intensity of the reward will be maximized as each subject chooses the reward that is most salient to him or her. In considering this problem, it may be that autonomic, facial, and/or verbal responding are not as pronounced as expected during the reward tasks. Yet, to assume that differences in intensity of emotion contributed to the lowered responsivity in the measured response systems, attenuated responding would need to be evidenced in all three subject groups.

Secondly, differences in baseline autonomic responding may exist between the RHD patients, LHD patients, and normal controls. This would make it difficult to separate deficits in baseline autonomic responding, per se, from affective autonomic responding. Consequently, baseline autonomic responding will be examined in the psychophysiological screening procedure.

Thirdly, it may be that the results of this study provide partial support for both the global and bivalent

models of emotion. For example, autonomic arousal (SCR and HR) may be mediated by the right hemisphere and hence RHD patients would show diminished responding during both shock and reward tasks. At the same time, facial activity, a more accurate index of valence, may provide support for the valence hypothesis such that RHD patients show reduced corrugator muscle activity during negative emotions, but accentuated zygomatic activity during positive emotion, whereas LHD patients would show the reverse pattern. The above is only one example of support for both the bivalent and global models. There are other possible outcomes indicating support for both models.

CHAPTER 4
RESULTS

First, analyses of the heart rate and skin conductance responding during the psychophysiological orienting procedure are presented. Next, primary analyses for Experiment 1 are presented for heart rate, skin conductance, ipsilateral corrugator EMG, bilateral zygomatic EMG, and verbal report ratings separately for the shock and reward conditions. Third, the analyses of the verbal report data from Experiment 2 are presented.

Following the analyses of group data, data from anterior and posterior subgroups and individual cases are examined.

Group Data

Psychophysiological Screening

To review, the orienting, or physiological screening procedure, consisted of an approximately 10 minutes period where the subjects were instructed to sit quietly and listen to tones. There were 8 block of three tones (24 tones total). Two of every three tones were 1000 hz and one was 400 hz. Heart rate and skin conductances responding was measured during the second before and six seconds following presentation of each tone. One subject was removed from the heart rate analyses due to unusually high and variable heart

rate. Additionally, one subject was removed from the skin conductance analysis due to faulty electrode connections.

Heart rate

Average heart rate change from baseline was examined using a Repeated Measures Analysis of Variance (ANOVA) with group (LHD, LH NCS, RHD, RH NCS) as the between subject factor and tone (low, high) as the within subject factor. The low tone was the novel tone. The main effect for group [$F(3,43) = .419$, $P = .740$], tone [$F(1,43) = 1.634$, $P = .208$], and the interaction between group and tone [$F(3,43) = .218$, $P = .884$] were all nonsignificant. See Table C-1 in Appendix C.

A repeated measures analysis of variance (ANOVA) was employed to examine D1 using group as the between subject factor (LHD, LH NCS, RHD, RH NCS) and tone (high, low) and block (1 to 8) as the within subject factors. Results revealed a main effect for tone [$F(1,43) = 8.63$, $P < .01$] such that there was a greater D1 for the low tone (the novel tone) compared to the high tone (the repeated tone). The mean D1 for the low tone was -3.4 (sd=4.40) bpm change from baseline whereas the mean D1 for the high tone was -2.5 (sd=3.82) bpm change from baseline. None of the other effects were significant. See Table C-2, the full ANOVA table, in Appendix C.

Skin conductance

The percentage of responses greater than .02 micro sieman was analyzed using a repeated measures analysis of variance with group (LHD, LH NCS, RHD, RH NCS) as the between subjects factor and tone (low and high) as the within subject factor. One RHD subject was excluded due to faulty electrode connections which resulted in corrupt data. Results revealed that the main effect of group, tone, and the group by tone interaction were not significant. The mean percentage of responses and standard deviations for each group were as follows: LHD, mean=8.07%, sd=25.73; LH NCS, mean=25.52%, sd=36.16; RHD, mean=7.67%, sd=19.19; RH NCS, mean=29.69%, sd=26.47. The full ANOVA table, Table C-3, is presented in Appendix C.

The recoded range corrected skin conductance response (SCR) was analyzed using a repeated measures analysis of variance (ANOVA) with group (LHD, LH NCS, RHD, RH NCS) as the between-subject factor and tone (low, high) and block (1 to 8) as the within subject factors. As mentioned above, one subject was excluded due to corrupt data. The main effect for group [$F(3,43) = 1.91$, $P = .1421$], block [$F(7,43) = 1.20$, $P = .3017$], and tone [$F(1,43) = .21$, $P = .6495$] were not significant. The interactions between block and group [$F(21, 301) = .70$, $P = .8305$], tone and group [$F(3,43) = .33$, $P = .80$], and between block, tone, and group [$F(21,301) = .87$, $P = .6244$] were also not significant. The full ANOVA

table, Table C-4 is presented in Appendix C. The means and standard deviations for each group collapsed across tone and block were: LHD mean=3.881, sd=13.690; LH NCS mean=14.691, sd=27.809, RHD mean=3.895, sd=14.528; RH NCS mean=13.549, sd=23.073.

To sum, during the psychophysiological screening procedure, subjects had a greater heart rate D1 to the novel tone. There were no differences between the tones in overall heart rate, percentage of SCR responses, or amount of skin conductance responding. Additionally, there were no group differences found for either heart rate or skin conductance.

Experiment 1

Experiment 1 consisted of two tasks (shock or reward). During each condition, heart rate, skin conductance, ipsilateral corrugator EMG, and bilateral zygomatic EMG were recorded during a three second baseline, tone onset, and a six second anticipation period. Within each task, the tone onset signaled either a stimulus or control trial. High tones always signaled stimulus trials (i.e., shock and reward) and low tones always signaled control trials. There were 40 trials within each task which were divided into four 10-trial blocks. Within each block there were 5 stimulus and 5 control trials. Subjects were administered the Self Assessment Manikin at the end of each 10-trial block.

Shock task

As mentioned above, subjects received the shock in the forearm ipsilateral to their lesions. Additionally, RH NCS and LH NCS received the shock on their right and left arms respectively. Subjects were asked to determine the level of shock that was "uncomfortable, but not painful." The level of shock chosen by the subjects was examined using a 1 factor ANOVA with group (LHD, LH NCS, RHD, RH NCS) as the between subject factor. There were no group differences in the voltage of shock chosen [F(3,44) = 1.79, P = .1622]. The means and standard deviations for each group in volts are as follows: LHD group (mean=68.75, sd=14.79), LH NCS (mean=57.08, sd=12.70), RHD group (mean=64.17, sd=12.58), RH NCS (mean=64.58, 9.40). The ANOVA table is presented below.

Table 4-1 ANOVA Table of Amount of Shock

	SS	DF	MS	F	Sig of F
Group	843.229	3	281.07	1.79	.1622
Residual	6893.750	44	156.67		

Heart rate. A series of separate analyses were conducted to examine several heart rate variables. These included overall heart rate change from baseline, D1 (the greatest deceleration within the first 3-seconds after tone offset), A1 (the greatest acceleration following D1 within the 6-second period), and D2 (the greatest deceleration

following A1 within the 6-second period). One subject was excluded from the LH NC group due to unusually high and variable heart rate. Figure C-1, C-2 and C-3 depict the heart rate wave forms in half second intervals for the NCS, RHD, and LHD subjects respectively.

Average heart rate change from baseline was examined using repeated measures analyses of variance (ANOVAs) for the shock condition with group as the between subjects factor (LHD, LH NCS, RHD, RH NCS) and condition (shock, no-shock) as the within subject factor. The analyses revealed no group differences [$F(3,43) = 1.55$, $P = .214$] as well as no differences between the shock and no-shock conditions [$F(1,43) = .050$, $P = .824$]. The interaction of group and condition was also not significant [$F(3,43) = .927$, $P = .436$]. The means for each group were as follows: LHD mean=-.558, sd=.985; LH NCS mean=-.130, sd=.650; RHD mean=.139, sd=1.556; RH NCS mean=-.121, sd=1.01. The complete ANOVA table is depicted in Table C-5 of Appendix C.

Heart rate D1 was examined using repeated measures analyses of variance ANOVAS) with group (LHD, LH NCS, RHD, RH NCS) as the between subject factor. The two within subject factors were block (1 to 4) and condition (shock and no-shock). Analysis of D1 revealed that there was a significant three way interaction between group, condition, and block [$F(9,43) = 2.09$, $P < 05$]. None of the other interactions or main effects were significant. The full

ANOVA table, Table C-6, is presented in Appendix C. To further explore the three way interaction, separate repeated measures analyses of variance (ANOVAS) for each condition with group as the between subject factor and block as the within subject factor were conducted. For the shock condition, the main effect for group, block, and the interaction were all nonsignificant. Examination of the no-shock condition, revealed that there was a main effect for group [$F(3,43) = 3.38$, $P < .05$] and a block by group interaction [$F(9,129) = 7.77$, $P < .05$]. The main effect for tone by block was not significant. See Tables C-7 and C-8 in Appendix C.

Post-hoc analyses of the group effect were conducted using independent t-tests with a Bonferroni correction. Because there were no significant differences between the RH NCS and the LH NCS [$T(1,21) = 1.753$, $P = .0942$], the control subjects were combined into one group and compared to the LHD and RHD subjects. Since three comparisons were made, the p-value needed to be $< .017$ to reach significance. The LHD group (mean=-2.30, sd=2.57) had a greater D1 compared to the RHD group (mean=-1.15, sd=1.58) during the no-shock condition [$T(1,22) = -2.605$, $P < .0162$]. There were no significant differences between the LHD group and the CONs or the RHD group and the CONs. The means and standard deviations for the CONs are reported below: LH NCS (mean=-

1.13, sd=1.45), RH NCS (mean=-1.94, sd=2.20). A table of the t-tests, Table C-9, is presented in Appendix C.

Post-hoc analyses of the group by block interaction were performed. T-tests with Bonferroni corrections were conducted to compare the groups separately for each block. As mentioned above, the LH NCS and the RH NCS were combined. Using the Bonferroni correction of $p < .017$ for significance, no significant differences between groups were revealed during blocks 1, 3, and 4. However, during block 2, the LHD subjects had a greater D1 [$T(1,22) = -2.62$, $P < .017$], (mean=-3.42, sd=2.64) compared with the RHD subjects (mean=-1.23, sd=1.16), but not the CONs [$T(1,33) = -2.10$, $P = .043$]. Tables of the t-tests for each block, Table C-10, C-11, C-12, and C-13, are presented in Appendix C. The means for each group for each block are presented below.

Table 4-2 Means and Standard Deviations of D1 during the Control Trials of the Shock Condition

	Block One	Block Two	Block Three	Block Four
LHD	-1.0583 (1.987)	-3.417 (2.638)	-3.050 (3.206)	-1.683 (1.716)
LH NCS	-1.718 (.745)	-1.063 (1.943)	-.945 (1.662)	-.800 (1.202)
RHD	-1.483 (1.206)	-.850 (1.141)	-1.875 (1.475)	-.4000 (2.072)
RH NCS	-2.342 (2.807)	-2.175 (2.406)	-1.025 (1.716)	-2.225 (1.644)

As with D1, a repeated measures ANOVA was conducted using group as the between-subjects factor and block and

condition was within-subject factors. There were no significant main effects or interactions. The overall means for each group are as follows: (LHD mean=1.70, sd=3.70; LH NCS mean=1.61, sd=2.88; RHD mean=2.02, sd=4.10; RH NCS mean=1.90, sd=3.47). The ANOVA tables, Table C-14, is presented in Appendix C.

The same type of repeated measures analyses with group as the between subjects factor and condition and block as the within subject factors was used to examine D2. Analysis of D2 revealed that there were no significant main effects. There was, however, a significant block by group interaction [$F(9,43) = 2.34$, $P < .05$] . See Table C-15 in Appendix C for details of the ANOVA table. The means for each group for each block are presented below.

Table 4-3 Means and Standard Deviations for D2 during the Shock Condition

	Block One	Block Two	Block Three	Block Four
LHD	-.779 (2.416)	-2.008 (3.1996)	-.638 (2.962)	.038 (2.172)
LH NCS	-.977 (1.499)	-.182 (1.362)	.123 (1.479)	-.114 (1.360)
RHD	.075 (1.824)	.179 (2.717)	-.629 (1.190)	-.833 (3.039)
RH NCS	-.633 (3.429)	-.650 (1.989)	-.446 (2.069)	-.465 (1.998)

To further examine the block by group interaction, separate ANOVAs were performed for each block. Group was the between subjects factor. There were no significant

differences between groups for block one [F(3,43) = .922, P =.438], block three [F(3,43) = .705, P = .554], and block four [F(3,43) = .945, P = .427]. However, a significant difference was found between the groups at block two [F(3,43) = 5.56, P < .01]. See the ANOVA tables in Appendix C, Tables C-16, C-17, C-18, and C-19.

Further exploration of the group differences during block 2, independent t-tests with a Bonferroni correction revealed that the LHD patients has a significantly lower D2 than the CONS [T(1,33) = -3.26, P <. 01] and RHD [T(1,22) = -3.27, P < .01 groups. The t-tests are presented in Table C-20 in Appendix C.

To sum, overall HR did not differ between the shock and control condition or between groups. Additionally, no significant group, condition, or block differences were revealed when examining A1. Examination of D1 revealed a three way interaction, that upon further exploration yielded no significant differences. One significant finding was revealed in D2. Specifically, there was a significantly greater D2 deceleration for the LHD group then the RHD group and LH NCS during block 2 only.

Skin conductance. Skin conductance was examined by exploring the percentage of responses (responses greater than .02 micro sieman) and recoded range corrected skin conductance response magnitude (responses that were greater than .02 micro sieman were corrected based on individual

responses and responses less than .02 were recoded to 0). One RHD subject was removed from the analyses due to corrupt data (the experimenter was unable to get the electrodes to remain firmly attached to the subject's palm).

Repeated measures analyses of variance were used with group as the between subject factor and condition (shock, no-shock) as the within subject factor. Block was not included in these analyses because there were not enough responses within each block. Results revealed a main effect for group [$F(3,43) = 3.13$, $P < .05$], a main effect for condition [$F(1,43) = 29.52$, $P < .001$], as well as an interaction between condition and group [$F(3,43) = 6.47$, $P < .01$]. See Table C-21 for the full ANOVA table in Appendix C.

The main effect of group was explored using independent t-tests with a Bonferroni correction. Since the difference between the LH NCS and RH NCS was not significant, these groups were combined. Three comparisons were conducted with a Bonferroni correction of $P < .017$. Results indicated that none of the groups were significantly different from one another. There was a lower percentage of responses in the RHD group (mean=13.18%, sd=17.15) compared to the CONS (mean=35.00%, sd=29.00), however, this difference did not reach significance [$T(1,34) = 2.304$, $P = .0276$]. The LHD group (mean=15.83%, sd=25.09) did not have a significantly

lower percentage of responses compared to the CONS or RHD groups. See Table C-22 in Appendix C.

The main effect for condition indicated that subjects had a greater percentage of SCR responses during the shock (mean=30.31%, sd=31.00) compared to the no-shock condition (mean=19.79, sd=25.43).

The condition by group interaction was explored using independent t-tests with Bonferroni corrections. Groups were compared to one another for both the shock and no-shock conditions. Since there were no significant differences between the two control groups during both the shock condition [T(1,22) = -1.375, P = .1830] and the no-shock condition [T(1,22) = -1.236, P = .2296], the LH NCS and RH NCS were combined into one group. There were three comparisons for each tone, the bonferroni correction changed the significance level to .05/3=.017. Examination of the group difference during the shock condition revealed that the RHD group (mean=15.00%, sd=16.88) had a lower number of responses above .02 micro sieman compared to the CONS (mean=44.17%, sd=33.27), [T(33) = 2.734, P <.017]. The LHD group (mean=16.25%, sd=23.94) also had fewer responses than the CONS [T(22) = -2.582, P <.017). Additionally, no significant group differences were found in percent of responding during the no-shock trials. The results of the t-tests are presented fully in Tables C-23 and C-24 in Appendix C.

Due to the large amount of variance in the percentage of responses between subjects, arcsin transformations were used and the data was reanalyzed. Using the transformed data, the main effect for group, main effect of tone, and the interaction remained significant.

Repeated Measures Analyses of Variance were used to analyze the recoded range corrected skin conductance responses (SCR). As mentioned in the data reduction section, the range corrected SCR was corrected by denoting the largest response for each subject as 100%. Each of the smaller responses for that subject was recoded as a percentage of the largest response. The data was recoded by changing all trials in which the actual SCR was less than .02 micro sieman to 0%. The between subjects factor was group (LH, LH NCS, RH, RH NCS) and the within subject factors were condition (shock and no-shock) and block (1 to 4).

Examination of the recoded range corrected skin conductance responses (SCR) for the anticipatory shock task revealed main effects for group [$F(3,43) = 2.99$, $P < .05$], block [$F(3,43) = 14.05$, $P < .001$], and condition [$F(1,43) = 23.36$, $P < .001$]. There were also significant interactions between condition and group [$F(3,43) = 6.60$, $P < .001$] and block and condition [$F(3,43) = 3.83$, $P < .05$]. The ANOVA table, Table C-25, is presented Appendix C.

The main effect for group was explored using independent sample t-tests with a Bonferroni correction. Since the difference between the LH NCS and the RH NCS was not significant [T(1,22) = -1.247, P = .2254], these group were combined. Using the Bonferroni correction of P < .017, as the significance level, the RHD group displayed significantly smaller SCRs compared to the CONS [T(1,33) = 2.60, P < .017], but not the LHD group. The means and standard deviations are as follows: RHD group (mean=5.08, sd=8.93), LHD group (mean=8.12, sd=14.31), CONS (mean=14.30, sd=11.14). A table of the t-tests, Table C-26, is presented in Appendix C.

The main effect of tone revealed that the high tone was associated with a significantly higher response (mean=13.98) than the low tone (mean=7.16).

The main effect of block was explored using paired t-tests with a Bonferroni correction based on six comparisons, P < .008. The results revealed that the SCR was greater during block 1 (mean=16.13, sd=17.39) compared to block 2 (mean=9.84, 13.96) [T(1,46) = 4.24, P < .001], block 3 (mean=8.08, sd=13.09) [T(1,46) = 4.57, P < .0001], and block 4 (mean=8.22, sd= 15.20) [T(1,46) = 4.30, P < .0001. Blocks 2 and 3, 2 and 4, and 3 and 4 were not significantly different from one another. Table C-27 in Appendix C contains the values from the t-tests.

The tone by block interaction was also examined using paired -t-tests with a Bonferroni correction based on four comparisons, requiring a $P < .0125$ for significance. The SCR for the shock no-shock (mean=21.80, sd=19.89) was significantly higher than the low tone (mean=10.46, sd=12.24) for blocks 1 [$T(1,46) = -4.655$, $P < .0001$], and block 4 (shock condition: mean=12.27, sd=19.02; no-shock condition: mean=4.17, sd=8.48), [$T(1,46) = -3.433$, $P < .01$], but not block 2 (shock condition: mean=12.21, sd=16.89; no-shock condition: mean=7.46, sd=9.86), [$T(1,46) = -2.264$, $P = .0284$] and block 3 (shock condition: mean=9.63, sd=14.39; no-shock condition: mean=6.54, sd=11.59), [$T(1,46) = -1.449$, $P = .1542$]. The t-tests are presented in Table C-28 in Appendix C.

The condition by group interaction was explored using t-tests with Bonferroni corrections separately for the shock and no-shock conditions. Since there were not significant differences between the LH NCS and the RH NCS during the no-no-shock condition [$T(22) = -.257$, $P = .7995$] or the shock condition [$T(22) = -1.338$, $p = .1946$], the two groups were combined into one control group. Using the bonferroni correction, the analyses had to reach a p-value of $.05/3 = .017$ to be considered significant. T-test tables are provided in Appendix C, Tables C-29 and C-30. During the shock anticipation, the RHD patients (mean=5.15, sd=8.55) had significantly smaller responses then the CONS

(mean=20.52, sd=16.03), [T=(1,33) = 3.10, P < .017]. The LHD group (mean=8.99, sd=13.13) was not significantly different from any of the other groups. The difference between the LHD group and the CONS approached significance [T(1,34) = -2.152, P = .0386], suggesting that the LHD group had less responding compared to the CONS during the shock condition.

Examination of the means and standard deviations of the SCR revealed large standard deviations compared to the means. Thus, the overall ANOVA was conducted with log transformed data. Using log transformation, the findings remained the same as above.

Since past studies have found a significant positive correlation between SCR and arousal ratings, the correlations between range corrected skin conductance magnitude and the arousal ratings during the shock condition was obtained. The results revealed a trend towards significance for the control subjects [R = .36, Beta = -4.66, T(1,22) = -1.80, P = .085]. The correlation between SCR and arousal was not significant for the LHD group [R = .11, Beta = 1.18, T(1,10) = .343, P = .7390] or the RHD group [R = .287, Beta = -1.04, T = -.899, P = .3923].

In sum, subjects had a greater percentage of responses in response to the shock tone than in response to the control tone. Also, the results revealed that the RHD and LHD subjects did not have significantly fewer responses than

the CONS. During the shock trials, however, the RHD and LHD group had fewer responses than the CONS, whereas there were no group differences during the no-shock trials. Subjects demonstrated greater magnitude of responding during the shock compared to the no shock condition. Also, there was a significantly greater magnitude during block 1, than blocks 2, 3, and 4. Additionally, the shock tone produced significantly greater magnitude of responding when compared to the no-shock tone during blocks 1 and 4. Also, during shock anticipation the RHD group had significantly smaller responses than the CONS, whereas the LHD group did not significantly differ from any of the other groups.

Facial electromyography (EMG). Ipsilateral corrugator EMG (CEMG), left zygomatic EMG (ZGL), and right zygomatic EMG (ZGR) were analyzed separately using change from baseline as the dependent variables. Repeated measures ANOVAs were employed. Group was the between subject factor (LHD, LH NCS, RHD, RH NCS). Block (one to four) and condition (high and low) were the within subject factors. Results of the analyses revealed no significant main effects or interactions for either CEMG or ZGR. The mean change scores by group for each variable are presented in the Table 4-4 below. The ANOVA tables, Table C-31 and C-32 are presented in Appendix C.

Table 4-4 Mean Change Scores and Standard Deviations for Facial EMG during the Shock Condition

	CEMG	ZGL	ZGR
LHD	.011 (.077)	-.009 (.111)	-.001 (.094)
LH NCS	.006 (.094)	.015 (.076)	-.005 (.107)
RHD	.031 (.175)	.010 (.115)	.013 (.100)
RH NCS	.008 (.102)	.023 (.097)	.013 (.107)

There was, however, an interaction between group and block for zygomatic left [$F(9,132) = 2.45$, $P < .01$]. The ANOVA table, Table C-33, is presented in Appendix C. The group by block interaction was further explored by examining block differences separately for each group. The was a significant difference between the groups for block 1 [$F(3,44) = 3.054$, $P < .05$], but not block 2 [$F(3,44) = 1.657$, $P = .1901$], block 3 [$F(3,44) = 1.543$, $P = .2167$], or block 4 [$F(3,44) = 1.743$, $P = .1720$]. The results for block analyses are provided in Tables C-34, C-35, C-36, and C-37.

Further exploration of the main effect of group which was significant in the analysis of block 1 was conducted using independent t-tests with a Bonferroni correction. Results of these analyses revealed that none of the groups were significantly different from one another. These results are depicted in Table C-38 of Appendix C. The means and standard deviations are as follows: LHD patients (mean=-.047, sd=.082), LH NCS (mean=.013, sd=.042), RH NCS (mean=.025, sd=.060), RHD (mean=.020, sd=.076).

Due to the large amount of variance found for CEMG, ZGL, and ZGR, log transformations were conducted. The findings were identical to the results obtained using the raw data.

In sum, there were no significant effects between the shock and control trials in ipsilateral corrugator EMG or bilateral zygomatic EMG. There was a significant group by block interaction for the left-sided zygomatic EMG variable. Further exploration of this finding, however, revealed no significant differences between the groups.

Self-assessment manikin. Since the self-report ratings of valence, arousal, and dominance were determined using a 5-point SAM ratings, nonparametric statistics were used to examine the differences by condition and by group. Wilcoxon Tests for paired samples were used to analyze the differences in ratings between the shock and no-shock trials. Kruskal-Wallis Tests were used to analyze group differences. The ratings at time 1 and time 2 were averaged together for the analyses.

For all three variables, (i.e., valence, arousal, and dominance) there was a significant difference in the ratings for the shock and control trials. Within the shock condition, subjects reported less pleasant feelings during the shock compared to the shock-control trials [Z = -5.61, P <.0001] (shock, mean=3.32, sd=1.29; shock-control, mean=1.29, sd=.579). The main effect for arousal [Z = -

5.09, P <.0001] revealed that on average subjects reported feeling more aroused during the shock compared to the shock-control trials (shock, mean=3.30, sd=1.28; shock-control, mean=4.65, sd=.754). There was also a main effect for dominance [Z = -5.03, P < .001]. Examination of means indicate that subjects reported feeling less in control during the shock compared to the shock-control trials (shock, mean=4.08, sd=1.18; shock-control, mean=4.67, sd=.804).

The Kruskal-Wallis Tests revealed that there were no group differences for ratings of valence, arousal, and dominance for both the shock and control trials. These results are presented in Table C-39 of Appendix C.

In sum, overall subjects reported that during shock anticipation they experienced more unpleasantness, more arousal, and less control than during the no-shock anticipation. Additionally, the RHD, LHD, and NCS groups did not differ in the intensity of their ratings in both the shock and no-shock conditions.

Reward task

As mentioned above, subjects were given the choice of receiving dollar bills, scratch-off Florida lottery tickets, or a combination of both dollars and tickets in this task. Subjects were instructed that each time they heard the reward tone, they would receive 1 dollar or 1 ticket (depending on what they chose). After each 10-trial block,

the experimenter brought the dollars or tickets into the chamber and placed them on the table in front of the subject. The subjects were instructed that at the end of the task, they could take their money or tickets from the table.

Heart rate. Overall heart rate was explored by calculating average heart rate change from baseline and employing a repeated measures analyses of variance (ANOVA) with group as the between subjects factor (LHD, LH NCS, RHD, RH NCS) and condition (reward, no-reward) as the within subject factor. Additionally, heart rate D1, A1, and D2 were examined also using repeated measures ANOVAs. In these analyses group was the between subject factor and block (1 to 4) and condition (reward and no-reward) were the within subject factors. One subject from the LH NC group was excluded for having unusually high and variable heart rate. Figures C-4, C-5, and C-6, in Appendix C, depict the heart rate change in beats per minute over half second intervals for the NCS, RHD, and LHD groups respectively.

Results of the ANOVA for overall heart rate revealed no significant main effects for groups [$F(3,43) = .762$, $P = .522$] or condition [$F(1,43) = .961$, $P = .332$]. The interaction was also not significant [$F(3,43) = .425$, $P = .736$]. The ANOVA table, Table C-40, is presented in Appendix C. The mean change from baseline in beats per minutes for each group is as follows: LHD=-.675, sd=1.82; LH

NCS=-.178, sd=.747; RHD= -.467, sd=.853; RH NCS= -.370, 1.021.

As mentioned above D1, A1, and D2 data were analyzed separately using repeated measures ANOVAs with group as the between subject factor and condition (reward, no-reward) and block (1 to 4) as the within subject factors. There were no significant main effects or interactions for any of the three variables. ANOVA tables for these three variables are in Appendix C, Tables C-41, C-42, and C-43. A table of means for each of the three variables by group is presented below.

Table 4-5 Means and Standard Deviations of D1, A1, and D2 for the Reward Condition

	D1	A1	D2
LHD	-2.46 (3.56)	1.71 (3.82)	-1.05 (3.83)
LH NCS	-1.36 (1.75)	1.06 (2.16)	-.32 (2.23)
RHD	-1.57 (2.24)	.95 (2.52)	-.69 (2.38)
RH NCS	-2.22 (2.90)	1.70 (3.87)	-1.27 (2.93)

To sum, overall heart rate along with D1, A1, and D2 were not significantly different between the reward and reward-control trials. Additionally, there were no group differences in overall heart rate or D1, A1, and D2.

Skin conductance. Similar to the shock task, percentage of SCR responses and recoded range corrected SCR were analyzed separately. Also, one RHD subject was excluded from analyses due to corrupt data.

The percentage of responses where the response was greater than .02 micro sieman was analyzed using a repeated measures analyses of variance (ANOVA) with group (RHD, RH NCS, LHD, LH NCS) as the between subject factor and tone (high, low) was the within subject factor. There was no main effect for group [$F(3,43) = .478$, $P = .700$] or for tone [$F(1,43) = .562$, $P = .457$]. The interaction between group and tone was also not significant [$F(3,43) = 1.86$, $P = .151$]. The mean number of responses for each group (in percentages) are as follows: LHD mean=15%, sd=23.45; LH NCS mean=14.38%, sd=27.75; RHD mean=10.23%, sd=15.00; RH NCS mean=20.63%, sd=15.13. See Table C-44 for the full ANOVA table in Appendix C. Arcsin transformations were also calculated and analyzed using the same design. The transformed data also revealed no significant results.

A repeated measures ANOVA was used to analyze the SCR. The between subjects factor was group (LH, LH NCS, RH, RH NCS) and the within subject factors were tone (high and low) and block (1 to 4). Examination of SCR revealed a main effect for block [$F(3,43) = 2.84$, $P < .05$] along with a block by condition interaction [$F(3,43) = 3.32$, $P < .05$]. The full ANOVA table, Table C-45, is presented in Appendix C.

Paired t-tests with a Bonferroni correction requiring $P < .008$ to reach significance were employed to explore the main effect of block. None of the block were significantly

different from one another, however, the difference between blocks 1 (mean=9.66, sd=13.07) and block 2 (mean=5.84, sd=11.64) approached significance [T(1,46) = 2.727, P = .009]. Block 3 (mean=6.17, sd=12.76) and Block 4 (mean=6.86, sd=14.82) were not significantly different from each other or Blocks 1 and 2. A table of these t-tests, Table C-46, is presented in Appendix C.

To examine the block by condition interaction, four paired t-tests were used with a Bonferroni correction requiring a P < .0125 to compare the reward and control tones for each block. Results revealed that at block 1, SCR was significantly greater [T(1,46) = 3.172, P < .01] following the no-reward tone (mean=12.50, sd=14.28) than following the reward tone (mean=6.82, sd=11.19). There were no significant differences between the reward and no-reward conditions for the remaining blocks (block 2: mean of no-reward=5.32, sd=10.82; mean of reward=6.36, sd=12.50, block 3: mean of no-reward=5.16, sd=11.62; mean of reward=7.18, sd=13.86, and block 4 mean of no-reward=6.79, sd=13.11; mean of reward=6.93, sd=16.50). The results of the t-tests are presented in Table C-47 in Appendix C.

The SCR magnitude was also examined using logarithmic transformations. Using the same design, the results were unchanged from the findings obtained using the nontransformed data.

In sum, there were no differences in the percentages of responses greater than .02 micro sieman for the reward versus control trials or between groups. In general, there were no significant differences in subjects responses during block 1, 2, 3, and 4. Additionally, responses were greater, however, after no-reward trials compared to the reward trials during block 1. There were no differences in the amount of responding between the reward and no-reward for blocks 2, 3, and 4. There were also no group differences detected in SCR magnitude.

Facial electromyography(EMG). Ipsilateral corrugator EMG (CEMG), left zygomatic EMG (ZGL), and right zygomatic EMG (ZGR) were analyzed separately. Repeated measures analyses of variance were employed. Group was the between subject factor (LHD, LH NCS, RHD, RH NCS), while block (one to four) and tone (high and low) were the within subject factors. There were no significant differences found for any of the facial muscle variables during reward condition. A means table reporting means for each variable by group is presented below. The three ANOVA tables, Tables C-48, C-49, and C-50, are presented in Appendix C. There were no significant main effects or interactions in the analyses of CEMG, ZGL, and ZGR. The ANOVAs were also employed using logarithmic transformations of the data. The transfomed data did not change the results in any way.

Table 4-6 Means and Standard Deviations of the Change Scores for Facial EMG during Reward Condition

	CEMG	ZGL	ZGR
LHD	.013 (.058)	-.034 (.093)	-.013 (.058)
LH NCS	.032 (.066)	.025 (.103)	.016 (.082)
RHD	.041 (.229)	.016 (.067)	-.015 (.123)
RH NCS	-.023 (.107)	.038 (.127)	.006 (.056)

Self-assessment manikin. As with the shock condition, the self-report of valence, arousal, and dominance were analyzed using nonparametric statistics.

Within the reward condition, Wilcoxon Tests for paired samples revealed that there was a significant difference between the reward and control trials for valence [$Z = -5.31$, $P < .0001$] which demonstrated that subjects reported more pleasant feelings during the reward (mean=1.25, sd=.562) compared to the reward-control (mean=2.90, sd=1.43) trials. There was also a main effect of tone for arousal [$Z = -2.71$, $P < .01$]. Subjects reported more arousal during reward (mean=3.74, sd=1.32) compared to the reward-control (mean=4.08, sd=1.08) trials. The main effect of tone for dominance [$Z = -2.52$, $P < .05$] revealed that subjects reported feeling more in control during the reward (mean=4.55, sd=.916) compared to the reward-control (mean=4.33, sd=1.07) trials.

Kruskal-Wallis Tests demonstrated that there were no group differences for any of the variables during both the

reward and control trials. See Table C-51 in Appendix C for details.

In sum, overall subjects reported greater pleasure, arousal, and dominance during the reward compared to the control trials. Additionally, none of the groups differed in their ratings during the reward or control trials.

Shock versus reward

In order to directly compare the change in emotional experience between the two stimulus and control conditions, the no-shock and no-reward control conditions were subtracted from their respective stimulus conditions. Thus, a new variable was created for each variable by subtracting the value during the shock condition from the value during the no-shock condition. Similar variables were created by subtracting the no-reward condition from the reward condition. These new variables were created in order to directly compare the change in each dependent variable between the stimulus and control condition in the shock task with the change between the stimulus and control condition in the reward task.

Heart rate. As mentioned above, overall heart rate along with D1, A1, and D2 were examined separately. Also, as in the heart rate analyses reported above, one subject was excluded from the LH NC group due to unusually high and variable heart rate change scores.

Direct comparison of the mean HR change during the shock and reward conditions was also examined by employing a repeated measures analyses of variance (ANOVA) using group (LH, LH NCS, RH, RH NCS) as the between subject factor and condition (shock minus no-shock and reward minus no-reward) as the within subject factor. There was no significant difference within the two tasks [$F(1,43) = .237$, $P = .629$] or between groups [$F(3,43) = 1.060$, $P = .376$]. The group by task interaction was also not significant [$F(3,43) = .400$, $P = .754$]. The mean bpm change from baseline for each group are as follows: LHD mean=.032, sd=2.02; LH NCS mean=.343, sd=.820; RHD mean=-.177, sd=2.012; RH NCS mean=.435, sd=1.439). The ANOVA table, Table C-52, is presented in Appendix C.

Heart rate D1, A1, and D2 values for shock task were compared to the values for reward task by subtracting the control values from the stimulus values for each subject for each trial block. These values were compared using a repeated measures analysis of variance for each variable. The between subject factor was group (LHD, LH NCS, RHD, RH NCS) and the within subject factor was task (shock minus shock-control and reward minus reward-control). There were no significant differences between the shock and reward conditions for any of the three variables. The ANOVA tables, Table C-53, C-54, C-55, are presented in Appendix C.

The means for each variable by group are reported in the table below.

Table 4-7 Group Means and Standard Deviations of D1, A1 and D2 comparing Shock and Reward Conditions

	D1	A1	D2
LHD	-.129 (3.809)	.211 (4.466)	.677 (4.415)
LH NCS	.232 (2.005)	.500 (2.628)	.320 (2.553)
RHD	-.2997 (3.401)	-.855 (4.654)	-.404 (3.684)
RH NCS	.269 (3.451)	.144 (4.649)	.183 (4.061)

To sum, there were no differences between the shock and reward tasks or group differences in overall heart rate. Also, there were no differences between groups, blocks, or tasks in heart rate D1, A1, and D2.

Skin conductance. The shock and reward tasks were directly compared by calculating new variables by subtracting the percentage of responses for control trials from the percentage of responses for respective stimuli trials (shock minus shock-control and reward minus reward-control). The shock and reward tasks were also directly compared to examine differences in recoded range corrected skin conductance responses (SCR). Again the no-shock and no-reward control trials were subtracted from their respective stimulus trials separately for each task and block. Also, one subject from the RHD group was excluded due to corrupt data.

Repeated measures analyses of variance were used to analyze these new variables. Group was again the between subject factor and task (shock minus shock-control and reward minus reward-control). The full ANOVA table, Table C-56, is presented in Appendix C.

The results revealed a main effect for group [$F(3,43) = 6.534$, $P < .01$ and task [$F(1,43) = 16.499$, $P < .001$]. The interaction of group by task approached significance [$F(3,43) = 2.814$, $P = .0504$].

Because the LH NCS and RH NCS were not significantly different from one another [$T(1,22) = -1.034$, $P = .3125$], these groups were combined. Exploration of the main effect of group using independent t-tests with a Bonferroni correction requiring a $P < .017$ for significance revealed that the difference between the percentage of responses between the control and stimulus trials was significantly smaller for both the LHD group (mean=1.25, sd=5.49) [$T(1,34) = -2.86$, $P < .01$] and the RHD group (mean=-.227, sd=3.25) [$T(1,33) = 3.28$, $P <.01$] compared to the CONS (mean=10.83, sd=10.88). There were no significant differences between the two patient groups [$T(1,21) = .776$, $P = .4465$]. A table of the t-tests, Table C-57, is presented in Appendix C.

The main effect of task revealed that subjects had a greater difference between the percentage of responses during the shock compared to shock-control trials

(mean=10.426, sd=15.102) than the reward compared to reward-control trials (mean=1.170, sd=9.957).

The interaction was explored using separate t-tests with Bonferroni correction for the shock and reward tasks. Since there were no significant differences between the LH NCS and the RH NCS during the shock task or the reward task, the groups were combined. Results revealed that during the shock task the LHD group, (mean=.833, sd=7.334) had a smaller difference between the percentage of responses for the shock and control trials compared to the CONS (mean=18.33, sd=16.73), [T(1,34) = -3.443, P < .01], but not the RHD group (mean=3.636, sd=5.95), [T(1,21) = -1.000, P = .3285]. The RHD group also had a significant smaller difference in the percentage of responses greater than .02 micro sieman than CONS [T(1,33) = 32.814, P < .01]. There were no significant differences between any of the groups for the reward minus reward control variable. Tables of the t-tests, Table C-58 and C-59, are presented in Appendix C.

A repeated measures analysis of variance (ANOVA) was conducted to compare SCR magnitude between the two tasks. Group (LHD, LH NCS, RHD, RH NCS) was the between subjects factor and task (shock minus no-shock and reward minus no-reward) and block (one to four) were the within subject factors.

There was a main effect for group [F(3,43) = 4.42, P < .01] along with a main effect for task [F(1,43) = 24.82, P <

.001]. There was also task by group interaction [F(3,43) = 4.81, P < .01] and a block by task interaction [F(3,43) = 6.81, P < .001]. The main effect for block, the interaction of block by group, and the three way interaction were all not significant. The results of the full ANOVA table are presented in Appendix C, Table C-60.

The main effect by group was further explored using independent t-tests with a Bonferroni correction. Since the difference between the LH NCS and RH NCS was not significant [T(1,22) = .509, P = .6159], these two groups were combined. Using the significance level of P < .017, based on the Bonferroni correction, the RHD group (mean=-.829, sd=2.61) had smaller SCR magnitude compared to the CONS (mean=6.212, sd=8.30), [T(1,33) = 2.733, P < .017]. There was no difference between the LHD group (mean=.481, sd=3.40) and the CONS or the LHD and the RHD. The results of the t-tests are presented in Table C-61 in Appendix C.

The main effect by task revealed that the shock condition produced significantly greater responding than the reward condition when the respective control conditions were held constant. The mean for the shock minus shock control variable was 6.82 (sd=15.71) whereas the mean for the reward minus reward control variable was -.62 (sd=13.06).

The block by task interaction was explored using paired t-tests with a Bonferroni correction requiring P < .0125 for significance. The results revealed that the SCR magnitude

during the shock task was significantly greater than the reward task for block 1 [T(1,46) = 4.724, P < .0001] and 4 [T(1,46) = 2.925, P < .01], but not 2 [T(1,46) = 1.59, P = .1195] and 3 [T(1,46) = .433, P = .6671]. A table of the t-tests, Table C-62, is presented in Appendix C. A means table is presented below.

Table 4-8 Means and Standard Deviations of Recoded Range-Corrected SCR Comparing Shock and Reward Tasks by Block

	Shock	Reward
Block One	11.35 (16.71)	-5.68 (12.28)
Block Two	4.75 (14.39)	1.04 (10.89)
Block Three	3.09 (14.62)	2.02 (11.57)
Block Four	8.10 (16.18)	.140 (15.91)

Independent t-tests with Bonferroni corrections were used to examine the task by group effect. Separate t-tests comparing groups were conducted for each task. Since there were no significant differences between the LH NCS and the RH NCS during shock task [T(1,22) = -1.209, P = .2393] or reward task [T(1,22) = .082, P = .9355], the two groups were combined. Examination of the shock condition revealed that the LHD group (mean=1.735, sd=6.545) had a significantly smaller difference between the shock and control trials compared to the CONS (mean=12.43, sd=12.29), [T(1,34) = -2.809, P < .01]. The RHD patients (mean=.127, sd=3.582) also significantly smaller differences between the shock and control trials when compared with the CONS, [T(1,33) = 3.234, P < .01]. The LHD and RHD groups did not differ from

one another [T(1,21) = .721, P = .4790]. There were no significant differences between any of the groups for the reward minus reward-control condition. Both tables of t-tests, Table C-63, and C-64, are presented in Appendix C.

To sum, subjects had a greater percentage of responses in anticipation of the shock compared to anticipation of reward. Additionally, both RHD and LHD patients had fewer responses than RH NCS, but not the LH NCS. Also, examination of magnitude of SCR revealed that subjects had greater magnitude of responding during the first and last trial block compared to the middle trial blocks. Additionally, both the LHD and RHD patients had significantly smaller SCRs compared to their respective controls during the shock task, whereas no group differences were revealed during the reward task.

Facial electromyography. Analyses were also performed to directly compared the shock and reward tasks. To do this new variables were created by subtracting the control trials from their respected stimulus trials for CEMG, ZGL, and ZGR. Separate repeated measures analyses of variance using group as the between subjects factor and task (shock minus no-shock and reward minus no-reward) as the within subject variable. The analyses revealed no significant effects for any of the three variables. The ANOVA tables, Tables C-65, C-66, and C-67, are reported in Appendix C. The means for

each group for CEMG, ZGL, and ZGR are presented in Tables 4-9, 4-10, and 4-11 below.

Table 4-9 Means and Standard Deviations of Corrugator EMG Comparing the Shock and Reward Tasks

	Shock	Reward
LHD	.004 (.108)	.004 (.138)
LH NCS	-.0061 (.084)	.015 (.104)
RHD	-.030 (.190)	-.072 (.565)
RH NCS	-.014 (.171)	-.031 (.163)

Table 4-10 Means and Standard Deviations of Left-Sided Zygomatic EMG Comparing the Shock and Reward Tasks

	Shock	Reward
LHD	.007 (.169)	.009 (.187)
LH NCS	-.013 (.084)	.028 (.106)
RHD	.009 (.184)	-.009 (.170)
RH NCS	.038 (.141)	.026 (.412)

Table 4-11 Means and Standard Deviations of Right-Sided Zygomatic EMG Comparing the Shock and Reward Tasks

	Shock	Reward
LHD	-.002 (.147)	-.019 (.147)
LH NCS	-.007 (.096)	.031 (.112)
RHD	-.006 (.156)	.003 (.110)
RH NCS	.048 (.159)	.005 (.193)

No main effects of group, block, or task or interactions were revealed for the analyses of CEMG, ZGL, and ZGR.

Self-assessment manikin. The SAM ratings for the shock and reward conditions were directly compared by creating new

variables. The new variables were calculated by subtracting the mean control rating from the mean stimulus rating within each task. Each variable was analyzed using Wilcoxon Tests for paired samples to explore differences in ratings by condition and Kruskal-Wallis Tests to examine group differences.

For all three variables there was a significant difference between the shock and reward tasks. The mean valence ratings [$Z = -5.69$, $P < .0001$] revealed that subjects rated the shock and reward tasks significantly differently. Examination of the means indicated that subjects reported feeling less pleasant during the shock than the control trials (mean=1.99, sd=1.30) and more pleasant during the reward than compared to the reward control trials (mean=-1.65, sd= 1.41).

Exploration of the means for the arousal ratings revealed that subjects reported greater arousal during the shock and reward conditions compared to their respective control conditions (shock mean=-1.34, sd=1.25; reward mean=-.344, sd=.923). The difference was significantly greater, however, between the shock and shock-control compared to the reward and reward-control [$Z = -4.16$, $P < .0001$].

The dominance ratings indicated that subjects reported feeling less in control during the shock than the no-shock trials (mean=-.583, sd=.947) and more in control during the

reward compared to no-reward trials (mean=.219, sd=.564), [Z = -3.95, P < .001].

There were no group differences in the ratings of valence, arousal, and dominance. See Table C-68 in Appendix C for details.

Subjects reported experiencing less pleasantness, more arousal, and less dominance during the shock compared to the reward task. There were no group differences in these ratings.

Medication effects

All medications that the subjects were taking at the time of the experiment were recorded. Groups differed in the amount of medications that affect the autonomic nervous system, including alpha and beta adrenergic blocking agents, calcium channel blockers, ACE inhibitors, and digoxin. Eleven of the RHD subjects were taking these medications, 9 of the LHD, 3 of the RH NCS and 4 of the LH NCS. Thus, the significant skin conductance analyses were reanalyzed using the presence of medications that affect the autonomic nervous system as a covariate.

Shock condition. Within the shock task, repeated measures analyses of covariance were conducted using percentage of responses with group as the between subject factor and condition (shock and no-shock) as the within subject factors. Presence or absence of medication was used as the covariate. When percentage of responses was examined

with the covariate, the main effect for group became nonsignificant [$F(1,42) = 2.04$, $P = .123$]. The condition by group interaction, however, remained significant [$F(3,43) = 6.47$, $P < .01$]. The full ANCOVA table, Table C-69, is presented in Appendix C.

Recoded range corrected SCR was also examined using medication as a covariate. In this analysis, group was the between subjects factor and block (1 to 4) and condition (shock and no-shock) were the within subject factors. Similar to the above analysis, using the covariate, the main effect of group lost it's significance [$F(3,42) = 1.57$, $P = .212$. Yet, again the condition by group interaction remained significant [$F(3,43) = 6.60$, $P < .01$]. This ANCOVA table, Table C-70, is also presented in Appendix C.

In sum, when medications that affect the autonomic nervous system (ANS) are used as a covariate, the group by condition interactions for both percentage of SCR responses and magnitude of SCR remains significant. As mentioned above, further exploration of this interaction revealed that the RHD and LHD groups had significantly fewer SCRs above .02 micro sieman than the RH NCS during the shock trials. Additionally, when the range corrected SCR values were examined, the RHD group had significantly smaller magnitude of responses than both controls groups, whereas the LHD group did not differ from any other groups.

Shock versus reward tasks. Since the results of the SCR data comparing the shock and reward conditions revealed significant results, medications were also used as a covariate in the analysis comparing the shock and reward trials. A repeated measures analyses of covariance was employed where group was the between subjects factor and task (shock minus no-shock and reward minus no-reward) was the within subject factor. When the percentage of responses was examined, the main effect for group remained significant [$F(3,43) = 5.25$, $P < .01$]. As in the ANOVA, the interaction by group and task approached significance [$F(3,43) = 2.81$, $P = .050$. The ANCOVA table, Table C-71, is presented in Appendix C.

A similar analysis was employed using recoded range corrected SCR as the dependent variable. In this analysis group was the between subjects factor and block (1 to 4) and task (shock minus no-shock reward minus no-reward) were the within subject factors. Again, the main effect for group remained significant [$F(3,42) = 3.82$, $P < .05$]. The interaction between group and task also remained significant [$F(3,43) = 4.81$, $P < .01$]. Table C-72, in Appendix C, depicts the results of this analysis. Appendix C.

Summary of medication effects. In sum, these analyses suggest that the presence of medications that affect the ANS does not account for the significant main effect of group for the percentage of SCR responses. As stated above,

exploration of this main effect revealed that the RHD and LHD groups had a significantly smaller difference between the stimulus and control trials compared to the RH NCS, but not the LH NCS. As in the ANOVA, the interaction between task and group approached significance.

The main effect of group and the group by task interaction for the magnitude of SCR remained significant. To review, the post-hoc analyses revealed that none of the groups significantly differed from one another in change in magnitude between the stimulus and control trials. The interaction of task and group, however, revealed that the LHD group had a significantly smaller difference between the shock and no-shock condition in SCR magnitude compared to the LH NCS. Also, the RHD group had a significantly smaller SCR magnitude compared to the RH NCS and LH NCS. There were no significant differences between the group when change in SCR magnitude between the reward and no-reward condition was examined.

Experiment 2

To review, Experiment 2 consisted of two conditions, shock and reward. Within each condition, there were two trials, stimulus and control. Each trial consisted of a five minute anticipation period during which the subjects were administered the Positive and Negative Affect Schedule and the Self Assessment Manikin. Subjects were instructed that at the end of the shock trial, they would receive one

shock of the same or greater intensity than the previous shocks. Additionally, subjects were instructed that at the end of the reward trial they would receive between 5 and 8 dollars or lottery tickets, which ever they chose. Also, they were informed that at the end of both 5 minute control trials, nothing would happen.

Shock condition

Positive and negative affect schedule. Repeated measures analyses of variance (ANOVAs) were used to explore positive affect factor (PA) and negative affect factor (NA) for both the shock and reward conditions. The between subject factor was group (LHD, LH NCS, RHD, RH NCS) and the within subject factor was condition (shock, control). There were no significant main effects or interaction for PA. However, a significant main effect for condition was revealed for NA [$F(1,44) = 9.52$, $P < .01$]. Specifically, subjects reported higher intensities of negative emotions during the shock (mean=12.42) compared to the shock-control trial (mean=11.42). The main effect of group or interaction between group and condition was not significant. These two ANOVA tables, Table C-73 and C-74, are presented in Appendix C.

Self-assessment manikin. Valence, arousal, and dominance ratings were analyzed using Wilcoxon and Kruskal-Wallis Tests. The shock and reward trials significantly differed for valence [$Z = -3.84$, $P < .001$], arousal [$Z = -$

3.44, P < .001], and dominance [Z = -2.55, P < .05]. Specifically, subjects reported less pleasant feelings during the shock trial (mean=2.60) compared to the control trial (mean=1.69), greater arousal during the shock (mean=3.88) compared to the control (mean=4.58). They also reported feeling less in control during the shock (mean=4.29) compared to control trial (mean=4.75).

There were no group differences in the valence and dominance ratings during both the shock and control trials. There was however, a significant group difference in arousal rating during the control trial, but not the shock trial. The Kruskal-Wallis Tests are presented in Table C-75 in Appendix C.

Mann-Whitney U Tests were used to examine the group effects. Both the LHD group (mean=4.833, sd=.577), [Z=-2.12, P < .05] and the RHD group (mean=4.92, sd=.289), [Z=-2.27, P < .05] reported significantly less arousal during the control condition compared to the LH NCS (mean=4.00, sd=1.35). The LHD and RHD group ratings were not significantly different from the RH NCS, (mean=4.58, sd=.515). The Mann-Whitney U tests are presented in in Table C-76 in Appendix C.

Summary of results of shock task. Subjects reported more negative affect, less pleasantness, more arousal, and less dominance during the shock compared to the no-shock condition. There were no differences in ratings of the

positive affect factor of the PANAS. Additionally, there were no group differences in reported in any variable, with the exception of the arousal ratings during the control condition. During the no-shock control condition the RHD and LHD groups reported significantly less arousal than the LH NCS, but not the RH NCS.

Reward condition

Positive and negative affect schedule. Repeated measures analyses of variance (ANOVAs) were used to explore positive affect factor (PA) and negative affect factor (NA) for the reward conditions. The between subject factor was group (LHD, LH NCS, RHD, RH NCS) and the within subject factor was condition (reward, no-reward control). Results revealed that there was a significant main effect of condition for PA [$F(1,44) = 7.52$ $P < .01$]. The mean score was 33.38 for the reward trial and 30.26 for the control trial indicating that subjects reported more positive affect during the reward compared to the reward-control trial. Examination of means revealed no significant main effects or interaction were found for NA. These two ANOVA tables, Table C-77 and C-78, are presented in Appendix C.

Self-assessment manikin. Valence, arousal, and dominance ratings were analyzed using Wilcoxon and Kruskal-Wallis Tests. Examination of the verbal report ratings of valence, arousal, and dominance during the reward and reward-control trials revealed a main effect for trial for

valence only [Z = -2.47, P < .05]. Arousal ratings were not significantly different between reward and control trials [Z = -1.83, P = .067]. Also, dominance ratings were not significantly different [Z = -1.15, P = .249].

There were also no significant group differences. See Table C-79 in Appendix C for details. Exploration of the main effect of condition for valence revealed that subjects reported feeling more pleasant during the reward (mean=1.23) compared to the reward-control trial (mean=1.62). The trend towards significance for arousal revealed that subjects reported feeling less calm during the reward (mean=4.21, sd=1.12) compared to the control (mean=4.53, sd=.997) trial.

Summary of results of reward task. In sum, subjects reported more positive affect and more pleasantness during the reward compared to the no-reward condition. There were no differences in ratings of negative affect, arousal, or dominance between the reward and no-reward conditions. Additionally, there were no group differences in the ratings of NA, PA, valence, arousal, or dominance.

Shock versus reward

Positive and negative affect schedule. The shock and reward condition were directly compared by creating new variables such that the control (no-shock and no-reward) ratings were subtracted from the respective stimulus (shock and reward) ratings. Repeated measures ANOVAs were conducted using group as the between subjects factor and

task (shock minus shock-control and reward minus reward-control) as the within subject factor. For both positive [$F(1,43) = 6.40$, $P < .05$] and negative affect factors [$F(1,43) = 8.64$, $P < .01$], there was a main effect of task. The main effect of group and task by group interactions were not significant for either PA or NA. The main effect of task for PA, revealed that subjects reported more positive affect during the reward compared to the no-reward condition (mean=3.13, sd=7.97) than the shock compared to the no-shock condition (mean=.298, sd=7.97). Examination of the mean differences between stimulus and control conditions for NA indicated that subjects reported more negative affect during shock compared to the no-shock condition (mean=1.00, sd=2.26) and slightly less negative affect during the reward compared to the no-reward condition (mean=-.085, sd=1.19). The ANOVA tables for both PA and NA, Table C-80 and C-81, are presented in Appendix C.

Self-assessment manikin. The shock and reward conditions were directly compared for each variable by creating new variables (shock minus no-shock and reward minus no-reward) for valence, arousal, and dominance. Wilcoxon Tests and Kruskal-Wallis Tests were performed to examine differences in condition and group respectively. The ratings for the shock condition were significantly different than the ratings for the reward condition for valence [$Z = -4.22$, $P < .0001$], arousal [-2.19, $P < .05$],

and dominance [Z = -2.13, P < .05]. Specifically, the ratings for the shock condition were more unpleasant than the shock control condition (mean=.936, sd=1.42), whereas the ratings for the reward trial were more pleasant than the reward-control trial (mean=-.383, sd=.968). Subjects also had higher ratings of arousal during shock compared to shock-control (mean=-.745, sd=1.170) than reward compared to reward control (mean=-.319, sd=1.218). Additionally, subject reported feeling more out of control during the shock compared to no-shock condition (mean=-.468, sd=1.14) than during the reward compared to no-reward condition (mean=-.085, sd=.503).

There were no group difference for valence, arousal, and dominance. See Table C-82 in Appendix C for details.

Summary of results comparing shock and reward tasks. Subjects reported a greater change between stimulus and control conditions indicative of more negative affect and less positive affect during shock task compared to reward task. Additionally, during the shock task, subject reported a greater change in valence, arousal, and dominance indicating more unpleasantness, more arousal, and less control compared to the difference between the reward and no-reward conditions. There were no group differences in ratings of PA, NA, valence, arousal, or dominance.

Subgroup Data

To better understand how specific lesion locations affect skin conductance responding, attempts were made to examine subgroups of the lesioned sample. Within the RHD group, there were 2 subjects with primarily anterior lesions, 3 subjects with posterior lesions, 5 subjects with mixed lesions, 1 subject with corrupt SCR data, and 1 subject whose CT scan could not be obtained, but a report of the scan stated that the patient had a temporal/parietal infarct. In the LHD group, 1 subject had an anterior lesion, 1 had a primarily anterior lesion, 6 had posterior lesions, 1 had a primarily posterior lesion, 3 had mixed lesions. Since dividing the groups into anterior, posterior, and mixed groups created groups that were too small for proper analysis, attempts were made to calculate prediction intervals and to examine individual differences in skin conductance responding. Unfortunately the prediction intervals were too large and included non-responders.

Tranel and Damasio (1994) found that RHD patients with attenuation or abolition of SCR when viewing emotional pictures had damage involving the right supramarginal gyrus and angular gyrus. Thus, subjects were divided into two new groups; anterior and posterior. Subjects in the anterior group had lesions that were previously considered anterior or mixed (not including areas 39 and 40). Subjects in the

posterior group had lesions that had been classified as posterior, primarily posterior, or mixed (including areas 39 and 40). Due to the small number of subjects within each group, descriptive information, rather than statistic analyses are presented. The descriptive information is depicted in Table 4-12.

Table 4-12 Means and Standard Deviations for Anterior and Posterior Groups during the Shock Task

	Lesion	%SCR		SCR	
		Shock	No-Shock	Shock	No-Shock
LHD	Pos. n=8	20.63 (27.83)	20.00 (32.07)	11.74 (17.91)	9.53 (14.49)
	Ant. n=4	7.50 (11.90)	6.25 (12.50)	3.49 (7.61)	2.71 (7.49)
RHD	Pos. n=6	6.25 (12.50)	3.75 (9.75)	2.95 (8.24)	2.37 (5.76)
	Ant. n=5	19.17 (19.34)	15.00 (23.24)	5.84 (6.92)	6.12 (10.71)
Both	Pos. n=14	15.83 (24.20)	14.58 (27.09)	8.81 (15.84)	7.14 (12.68)
	Ant. n=9	14.50 (17.07)	11.50 (19.30)	3.97 (6.99)	2.60 (7.05)

Percentage of Responses

LHD subjects with posterior lesions appeared to have a greater number of SCR responses. The reverse trend was observed in the RHD group such that patients with anterior lesions had a greater percentage of SCRs compared to patients with posterior lesions. Of note, relative to the differences between the anterior versus posterior groups

within both the LHD and RHD patients, the differences between the shock and no-shock conditions are very small. When subjects were divided into anterior versus posterior lesions regardless of side of lesion, the percentage of responses were almost identical.

SCR Magnitude

Similar to the trends observed for percentage of SCRs, the magnitude of SCRs was greater in LHD patients with anterior lesion compared to LHD patients with posterior lesions. Again, the opposite trend was observed in the RHD patients. Also, as noted in the examination of percentage of SCRs, the relative to the anterior/posterior differences, the differences between the shock and no-shock conditions is quite small. When the anterior and posterior groups of RHD and LHD patients were combined, the posterior group had a greater magnitude of response.

The Effect of Neglect

To examine the effect of neglect on SCR magnitude during the shock task, the SCRs of the right hemisphere subjects with neglect and/or extinction (n=5) were compared to the LHD and CONs. Similar to the overall findings, the LHD [$T(1,34) = -2.15$, $P < .05$, (mean=8.99, sd=13.13)] and the RHD subjects with neglect [$T(1,27) = 2.07$, $P < .05$, (5.32, sd=5.45)] had significantly smaller SCRs during the shock condition compared to the CONs (mean=20.52, sd=16.03). Also consistent with the overall findings, there were no

significant differences between the RHD and LHD groups [T(1,15) = .595, P = .5609].

The SCRs of the RHD subjects with clear neglect (n=3), excluding the subjects with evidence of extinction only, were compared to the LHD group and the CONs. In this analysis, the RHD group was not significantly different from the CONs [T(1,25) = 1.99, P = .0566], although the difference approached significance. However, the mean for the RHD group with neglect was extremely small (mean=1.667, sd=2.887) compared to the overall mean of the RHD subjects (mean=5.15, sd=4.56), suggesting that subjects with neglect demonstrate a greater impairment in SCR responding in anticipation of shock. As will be described below, two of the three subjects with neglect were non-responders. Since the number of subjects with neglect is quite small, however, the above findings need to be interpreted with caution.

Individual Case Studies

To examine individual differences in SCR and the dissociation between SCR and verbal report, each subjects percentage of SCRs and magnitude of SCRs during the shock and no-shock conditions, along with verbal report change scores are presented in Table 4-13 and 4-14.

Non-Responders

Within the RHD group, 36% of the subjects (4/11) were non-responders. Of the 4 non responders in the RHD group, 3 had lesions involving the supramarginal gyrus and angular

gyrus (areas 40 and 39, respectively). The fourth non-responder, R11, did not have a lesion involving areas 39 and 40. R11, however, reported only a small change (1 point total on valence, arousal, and dominance) in emotional experience during the shock compared to the control condition. No clear pattern of neurological impairment was apparent in the 4/12 (33%) of LHD subjects who were non-responders.

Percentage of SCRs

Of the RHD subjects who had responses, all but one, R14, demonstrated a greater percentage of responses during the shock compared to the no-shock condition. Although R14 did not demonstrate greater SCRs during shock compared to the no-shock condition, he reported increased unpleasantness, arousal, and loss of control during shock compared to the no-shock control.

Within the LHD group, 5 subjects who were responders, displayed a greater percentage of responses during the shock compared to the no-shock conditions. One LHD subject had the same percentage of responses during the shock compared to no-shock trials and two subjects had a greater number of responses during the no-shock compared to the shock trials. All three of these subjects who did not display the expected difference in the percentage of SCRs during shock compared to the no-shock condition reported the expected changes in emotional experience.

SCR Magnitude

Within the RHD group 2 subjects showed a clearly greater SCR magnitude during the shock compared to the no-shock control (greater than 1% point). Three subjects showed a small difference in the expected direction (less than 1%) and one subject had greater responding during the no-shock condition. Of the four subjects who did not display greater SCR magnitude in the shock compared to the shock control conditions, three subjects displayed the expected change in verbal report and one did not.

In the LHD group, five of the subjects who were responders demonstrated greater SCRs during the shock compared to the no-shock conditions. Three subjects did not have greater responding during the shock compared to the no-shock condition. One of these subjects, L12, also had a relatively small change in verbal report ratings.

Verbal Report

Three of the RHD subjects had no or minimal change in verbal report ratings (1 point or less between the shock and control conditions for valence, arousal, and dominance combined). Of those three subjects, 2 were non-responders. One subject who had a minimal change in verbal report ratings, displayed SCRs, but did not display a greater percentage or magnitude of SCRs in the shock compared to the no-shock conditions.

Within the LHD group, one subject had a minimal change in rated emotional experience (1 point change in combined valence, arousal, and dominance). This subject also was one of the non-responders.

Subject L7

Examination of the LHD subjects revealed that one individual, L7, had much greater percentage of SCRs and higher SCR magnitude compared to the other LHD subjects. L7 was the youngest of the LHD group. L7 was also the subject who had SCR measured on the right hand because his left arm had been amputated. As mentioned above, it was decided to include this subject in the analyses because recent evidence suggests that there are no differences in bilateral SCR measurements in brain damaged subjects (Tranel & Damasio, 1994).

To examine the influence of L7 on the overall SCR magnitude analyses, L7 was removed and the magnitude analyses were conducted again. T-tests were used to explore the tone by group interaction, using a Bonferroni correction of $p < .008$. The results revealed that along with the RHD group, the LHD group also had significantly smaller responding than the LH NCS [$T(1,21) = -3.43$, $P < .008$] and RH NCS [$T(1,21) = -3.08$, $P < .008$] during the shock condition. As in the above analyses, there are no significant group differences during the no-shock condition. Thus, when L7 is removed from the analyses, the LHD group

along with the RHD group displays an impairment in SCR magnitude during the shock condition.

Table 4-13 Comparison of SCR and Verbal Report in RHD Patients

	AGE	Shk %SCR	No-S %SCR	Shk SCR	No-S SCR	Val	Aro	Dom	Lesion
R 1	73	5	0	0.00	0.00	3	.5	1	Mixed 39,40
R 2	73	5	0	5.00	0.00	1	-2	-.5	Mixed
R 3	74	0	0	0.00	0.00	0	-.5	.5	Pos.
R 4	64	20	5	5.53	5.00	2	-1	.5	P.Ant.
R 6		25	15	11.80	9.46	3.5	-4	-1.5	Pos.
R 7	76	15	5	7.23	5.00	4	0	.5	Mixed
R 8	64	0	0	0.00	0.00	1	-2	-1.5	Pos.
R 11	57	0	0	0.00	0.00	0	-1	0	P.Ant.
R 12	48	25	20	9.81	9.07	2	-.5	0	No Scan
R 13	65	20	20	6.75	6.59	0	0	0	Mixed 39,40
R 14	49	55	60	10.54	20.14	3	-3	2	Mixed 39,40

Note: Shk=shock condition; No-S=No-shock condition; Val=valence; Aro=arousal; Dom=dominance; (P.Ant)=primarily anterior; (Pos.)=posterior; (39,40)=mixed involving areas 39 and 40

Table 4-14 Comparison of SCR and Verbal Report in LHD patients

	AGE	Shk %SCR	No-S %SCR	Shk SCR	No-S SCR	Val	Aro	Dom	Lesion
L8	72	0	0	0.00	0.00	1.5	0	0	Pos.
L3	76	10	•	8.23	1.90	2	0	-1	Pos.
L4	67	25	•	13.99	18.42	4	-1	0	Pos.
L9	60	0	0	0.00	0.00	1	0	0	Mixed 39,40
L6	68	25	25	8.98	10.84	3	-2.5	-1	Mixed
L7	50	85	95	47.85	33.72	2	-1.5	0	Pos.
L8	72	25	15	10.95	2.92	1.5	-1	0	Pos.
L9	60	0	0	0.00	0.00	3	-3	-3	Ant.
L10	68	5	0	5.00	0.00	2	-2	-2	Pos.
L11	76	0	0	0.00	0.00	3	-2.5	-1	P.Ant
L12	70	15	30	7.84	19.24	2	0	0	P.Pos
L13	62	5	0	5.00	0.00	3.5	-2	0	Mixed

Note: Shk=shock condition; No-S=No-shock condition; Val=valence; Aro=arousal; Dom=dominance; (P.Ant)=primarily anterior; (Pos.)=posterior; (39,40)=mixed involving areas 39 and 40

Table 4-15 SCR and Verbal Report in NC Subjects

	Shk %SCR	No-S %SCR	Shk SCR	No-S SCR	Val	Aro	Dom
RC1	85	50	48.31	15.05	3.5	-3.5	-1.5
RC2	10	5	8.87	1.43	2	-1	-1
RC3	0	0	0.00	0.00	1.5	-.5	0
RC4	45	40	9.97	14.89	3	-3.5	-3.5
RC5	95	55	42.17	16.63	1	1	0
RC6	100	40	61.58	9.13	4	-3.5	-3
RC7	45	40	16.56	7.66	3	-2.5	-1.5
RC8	95	80	49.11	27.22	0	-2	0
RC9	45	25	16.27	10.67	3.5	-2.5	-.5
RC10	25	5	11.12	.50	3	0	-1
RC11	70	35	26.92	7.89	.5	0	0
RC12	25	15	7.08	1.58	.5	.5	0
LC1	15	10	10.11	4.78	2.5	-2.5	0
LC2	100	100	39.62	30.42	4	-3.5	0
LC3	10	0	8.81	0.00	3.5	-2.5	-2
LC4	70	25	18.45	8.18	2	-2	-.5
LC5	30	20	13.89	9.26	0	0	.5
LC6	60	45	14.34	11.60	2	-.5	0
LC7	50	5	23.02	1.32	0	0	0
LC8	5	0	10.95	2.92	3	-3.5	0
LC9	40	20	20.54	7.41	1	-1	0
LC10	25	0	20.50	0.00	-.5	0	0
LC11	15	5	9.32	2.26	2.5	0	0
LC12	0	0	5.00	3.29	.5	0	-1

CHAPTER 5
DISCUSSION

In this study, emotional experience was measured in individuals with unilateral cortical strokes and individuals who were neurologically normal. Emotional experiences were evoked by in vivo unpleasant and pleasant anticipatory situations. Emotional responding was measured using verbal report, autonomic responding, and facial muscle activity.

This study is unique for several reasons. First, attempts were made to examine emotional experience in both negative and positive emotional situations. In other studies, when emotional experience has been examined in stroke patients, only unpleasant emotionally-evoking stimuli have been used. Using both pleasant and unpleasant situations made it possibly to explore the differences in predicted responding based on the global right hemisphere model of emotions and the bivalent model of emotions.

Second, in most of the other studies of emotional experience in stroke patients, emotions have been elicited using stimuli that require perceptual interpretation (i.e., emotional slides). In this study, an in vivo elicitation of emotion was used so that subjects did not have to make perceptual interpretations in order to comprehend the

emotional content of the situation. Thus, the subjects' abilities to comprehend the emotional context of these in vivo situations is not confounded with the perceptual problems that are common in patients with RHD.

Third, multiple response systems of emotion were examined in the present study. Specifically, skin conductance responding was used because it seems to be sensitive to emotional arousal (Greenwald, et al., 1989). Corrugator and Zygomatic EMG were examined because they have found to be useful indicators of emotional valence (Greenwald, et al., 1989). Additionally, heart rate was examined because it has been found to be useful in the study of anticipation (Lang et al., 1978). By using multiple response systems, the presence of potential differential breakdown, (i.e., dissociation between verbal report and autonomic responding), could be examined following hemispheric stroke.

Before discussing the manner by which hemispheric strokes affected emotional experience in this study, the data from the normal subjects is discussed. It is important to present the data on the normal subjects first to insure that the tasks produced data that fits with the current knowledge base regarding the psychophysiology of emotion. The summary of the findings based on the differential responding in the normal subjects is presented below,

followed by a discussion of the results with the stroke patients.

Differential Responding in Normal Subjects

Shock Condition

Heart rate

In the normal subjects, heart rate was expected to be greater during the shock compared to the no-shock condition. Additionally, a heart rate wave form, with an initial deceleration, followed by an acceleration, and then a second deceleration was expected during the shock condition. This wave form was expected to be attenuated during the no-shock control trials.

Heart rate did not differentiate the shock from the control trials within the normal controls. There are several possible explanations for the lack of significance between the shock and control trials.

First, heart rate tends to differ depending on the response-set of the subjects. For example, heart rate wave forms have been found to be much more pronounced when subjects are supposed to respond in some way at the end of the anticipation period (Lang, Ohman, Simons, 1978). In a recent study, when subjects were asked to react to a noxious noise which followed a 6 second warning cue, they had greater heart rate decelerations during the anticipation period than subjects who were not asked to respond in any way (Patrick & Berthot, 1995). This study, however,

differed from the present study in that subjects were not given differential cues to predict whether the noxious stimulus would occur or not.

Additionally, individual differences in heart rate responding during fear conditioning have been observed (Hare, 1972; Hodes, Cook, and Lang, 1985). While most individuals responded to fear anticipation with predominantly heart rate acceleration, some individuals have been found to respond to anticipation of a feared stimulus with heart rate deceleration. As a consequence, it may be that a larger sample size is needed for a significant heart rate acceleration to be observed statistically.

Skin conductance

The predictions that SCR would be greater during shock compared to the shock control trials were supported. Consistent with previous literature, skin conductance responses were greater during the threat of shock compared to safe trials (Bankart & Elliot, 1974; Bowers, 1971a, 1971b). As expected, SCR also habituated during the shock conditions, such that SCR during block 1 was significantly greater than during blocks 2, 3, and 4.

Facial electromyography

Corrugator EMG was expected to be greater during shock compared to the no-shock condition, whereas zygomatic EMG was expected to show a either a decrease or smaller increase during the shock compared to the no-shock trials. Other

studies have found that corrugator EMG is related to unpleasant emotional experience and zygomatic EMG is related to pleasant emotional experience (i.e., Greenwald, et al., 1989).

Ipsilateral corrugator and bilateral zygomatic EMG did not differentiate the shock from control trials. There are at least two possible factors that may have contributed to the lack of the expected finding: the age of the subject and the gender.

A recent study examining the relationship between age in the general population on surface EMG of pericranial muscles provides partial support for the decrease in EMG with age. Jensen and Fuglsang-Fredriksen (1994) revealed that EMG activity was significantly decreased in older individuals during maximal voluntary contraction. These authors suggest that the decrease in amplitude is related to decrease in number of muscle fibers along with an increase in age-related type II atrophy. However, in this study when subjects were exposed to pain (blood being drawn) and a cold-pressor test the increase in muscle activity was not affected by age. This study differs from the present in that different facial muscles were measured and that they were measured under voluntary contraction and exposure to pain. Moreover, the subjects in this study were divided into four age groups. The oldest group ranged from 55-64 years of age. In the present study, the average age is in

the mid sixties and many of the subjects over 70 years of age. It is possibly that age produces a greater decrease in EMG amplitude in subjects over 65.

Additionally, sex differences may have played a role in the lack of significant EMG findings. Females have been found to generate facial EMG of greater amplitude during affective imagery, show a stronger correlation between ratings of emotional experience and facial EMG, and demonstrate greater facial EMG during voluntary facial expression as compared to males (Schwartz, Brown, & Ahern, 1980; Dimberg & Lundquist, 1990). Since all but two subjects in this study were males, the facial EMG changes may be attenuated compared to findings in a mixed gender or female sample.

Verbal report

The differences in ratings of emotional experience revealed that, as predicted, subjects reported more unpleasantness, more arousal, and less control during the shock compared with shock-control trials. While the underlying emotional state of the subjects can not be inferred with certainty from their ratings, the appropriateness of the ratings illustrates that the subjects were able to accurately perceive the expected emotional tone of the situation when asked about the situation at a later time.

Additionally, during the shock task of Experiment 2 subjects reported significantly more negative affect during the shock compared to control trials, along with greater unpleasantness, arousal, and loss of control. As in Experiment 1, these rating support the predictions regarding this task. There were no differences in the positive affect ratings. These findings reflects subjects ability to accurately perceive the emotional context during the anticipatory period.

Reward Condition

Heart rate

Heart rate was predicted to produce a triphasic curve, including an initial deceleration, followed by an acceleration, and then a second deceleration during the reward compared to the no-reward trials. No significant differences, however, were found when the heart rate variables were examined during the reward condition.

As mentioned above, heart rate varies as a function of the response-set given to the subjects. This finding has been clearly demonstrated in anticipation of high interest slides (nude female) in a sample of undergraduate males. Simons, Ohman and Lang's (1979) subjects were divided into two groups. Each group was presented with two tones. One tone signaled the presentation of high interest slides, whereas the other tone signaled presentation of low interest slides. One group was instructed to press a switch, as

quickly as possible, following the presentation of the tone to insure that the slide would be presented for a full 5 seconds. The other group was told to just pay attention to the tones and the slides. The subjects who were given a response-set to react to had larger overall responses and greater deceleration than the group that did not have to react to the tone. Although the subjects were not asked to rate their emotional experience, the high interest slides are likely to be somewhat comparable to the anticipation of reward in the present study. In both studies, subjects are anticipating something with positive rather than negative or neutral valence.

Skin conductance responding

Unexpectedly subjects had greater responding during the reward-control trials compared to the reward trials. The meaning of this finding is unclear. One possible explanation is that subjects experienced the no-reward trials as "frustrative nonreward." Fowles (1988) and Tranel (1983) conceptualized the electrodermal system as an anxiety system that is influenced by punishment or frustrative nonreward. Since the subjects in this experiment are expecting to obtain dollars or lottery tickets as part of this task, the no-reward condition in the reward task may be experienced by the subjects as a frustrative non-reward situation. Specifically, perhaps the higher SCRs during the no-reward trials is related to subject's feeling

disappointed relative to the reward trials. Verbal report ratings illustrated that subjects felt less pleasant and less in control during the no-reward compared to the reward trials.

A second explanation for the lack of increased SCRs during the reward compared to the no-reward trials is related to the arousal level of the subjects. As mentioned in the literature review, SCR is highly correlated with arousal ratings (Greenwald, Cook, & Lang, 1989). Also, as mentioned in the design issues section of the literature review, one of the concerns about the reward task was that it was not as arousing as the shock task. Comparison of arousal ratings between the shock and reward conditions reveal that, in fact, subjects rated the shock condition as more arousing than the reward condition. As a consequence, it is possible that lack of increased SCRs during the reward compared to the reward-control trials is the result of the lack of arousal during the reward condition.

Moreover, Simons, Ohman, and Lang (1979) found that subjects' SCR did not differ during anticipation of high interest and low interest when subjects were not asked to respond motorically. In this experiment subjects are not asked to respond in any way following the anticipatory period. Thus, the lack of SCRs during the reward compared to the no-reward trials may be related to the lack of a motoric response-set.

Facial electromyography

Similar to the shock condition, none of the facial muscle sites, ipsilateral corrugator and bilateral zygomatic, differentiated between the reward and control trials. The possible reasons for the lack of findings during the reward task are the same possibly explanations for the lack of findings during the shock task; age and gender. These reasons that age and gender possibly contributed to the lack of significant results are discussed above.

Verbal report ratings

Subjects also reported more pleasantness, arousal, and greater feelings of control during the reward anticipation than the reward-control. Similar to the shock situation, this illustrates that the subjects are able to perceive the emotional tone of the situation accurately.

Additionally, in Experiment 2, subjects reported more positive affect, and more pleasantness during the reward compared to the control trials. There were no differences in reported negative affect, arousal, and dominance. These results reveal that subjects perceive the reward situation as more positive and pleasant than the control trials.

The negative findings of the arousal and dominance rating in Experiment 2 suggest that subjects were not as emotionally aroused or as in control during the reward condition in the second experiment compared the no-reward

control. These findings refute the predictions made about the emotional content of Experiment 2. Perhaps, since the subjects had already obtained a significant amount of dollars or lottery tickets (20 dollars or ticket) in Experiment 1, they were not as emotionally excited during Experiment 2.

Another possible explanation could be related to the conservative nonparametric statistics used for analyses. The Wilcoxon Test is a conservative test when used with only one rating, due to the large number of ties in subject ratings.

Group Differences in Emotional Responding

As presented above, for the most part, heart rate and facial EMG did not differentiate the shock and reward trials from their respective controls. The only significant group difference revealed in the heart rate analyses was that during the shock condition that LHD group had significantly greater decelerations during the control trials for block 2 of D2. These findings are of trivial theoretical importance. The discussion below will focus on the SCR and verbal report ratings.

During the shock condition, RHD subjects had smaller SCR than their respective controls. This replicates previous findings (Meadows & Kaplan, 1994; Zoccolotti et al., 1982; Heilman, et al., 1978). This finding is supportive of both the global and bivalent theory of

emotion. According to the global theory of emotion, RHD subjects are expected to display impairment in emotional processing of all types, whereas according to the bivalent view, RHD patients display a deficit in processing emotional content with a negative or unpleasant valence. Thus, the overall finding that RHD patients in this study have decreased responding during the shock condition is supportive of both theories.

However, the RHD and LHD subjects did not differ statistically from one another during the shock condition. This finding is inconsistent with both the global and bivalent views of emotion. Additionally, this finding is also contradictory with previous literature (i.e., Heilman et al., 1978). In previous studies, emotional slides (i.e., Zoccolitti et al., 1982; Meadows & Kaplan, 1994) and pain (Heilman et al., 1978) have been used in the past to elcit emotion. The present study differs in the use of an anticipatory paradigm.

Some of the above studies have found that LHD patients are hyperaroused and show increased SCR in response to unpleasant emotional experience (Heilman, et al., 1978). In this study LHD patients had SCRs that were smaller during the shock condition, but not significantly different from the control subjects. This replicates previous findings (Morrow et al., 1981; Meadwos and Kaplan, 1994).

No group differences were found on the verbal report ratings. It is interesting that although the RHD patients do not have a normal SCR while anticipating an electric shock, they nonetheless reported feeling the same intensity of unpleasantness, arousal, and loss of control as the LHD group and NCS. Meadows and Kaplan (1994) found similar results when measuring SCR and verbal report in RHD and LHD patient groups as they viewed emotional slides.

At this point, however, it is important to note that one LHD subject, L7, had a larger magnitude of SCRs than the other LHD subjects. When this subject is removed from the analyses, the LHD subjects have significantly lower SCR magnitude compared to NCs. This subject was unique in that SCR was measured from his hand contralateral to his lesion because his left arm had been amputated due to thromophebitis, a type of disease that cause blood clots within the peripheral veins. As mentioned above, recent evidence suggests that SCRs are not significantly different when measured on the left and rights hands of patients with brain damage (Tranel and Damasio, 1994). At the same time, it is important to restate that without inclusion of this subject, similar to the RHD group, the LHD group has significantly smaller SCR magnitudes compared to the NCs.

There are a few possible explanations for the decreased number and magnitude of SCRs during the shock condition in RHD and most LHD subjects. First, although the brain damage

subjects, along with the normal controls reported the expected changes in verbal report of emotion, it can not be assumed that subjects perceived the emotional situation accurately. At the end of each 10-trial block, the experiment asked the subjects to rate their experiences by asking "When you heard the high tone, and you knew you were going to get a shock, how did you feel on this scale..." Using this method, it is unclear whether subjects reporting their actual subjective experiences during the situation or whether they were reported what they are "expected" to feel.

Moreover, since the subjects were not required to respond in any way, it is unclear whether they were able to distinguish each tone on a trial by trial basis. However, all subjects were able to distinguish the pairs of tones before the onset of the experimental trials. Since subjects were not required to respond in any way to insure that they interpreted each anticipatory trial accurately, it is unclear whether the LHD and RHD subjects clearly understood the emotional context of the shock condition and reward conditions. As a consequence, the decrease SCRs in the RHD group and in most of the subjects from the LHD group, may be reflective of their inability to perceive the situation accurately on a trial by trial basis.

A second possiblity is that brain damage, in general, causes a decrease in SCR during expected emotional arousal. This explanation is unlikely in light of the previous

research conducted with LHD and RHD subjects (i.e., Heilman, et al., 1978; Meadows & Kaplan, 1994; Zoccolotti et al., 1982). Additionally, recent evidence by Tranel and Damasio (1994) suggests that certain regions of the brain within the left and right hemisphere affect SCR whereas other regions do not. There findings are discussed more fully below.

Global versus Bivalent Models of Emotion

Subjects displayed differential SCRs in the shock compared to the no-shock conditions. RHD patients, however, showed a paucity of responding when their SCR magnitude was compared to the NCs. This finding is consistent with both the global and bivalent theories of emotion. Both of these theories predict that RHD will cause a deficit in emotional processing of unpleasant or negative emotional states. Valence effects during the reward condition were needed to provide overall support for the global or bivalent models. According to the global theory of emotion, RHD patient would show deficiencies in the emotional experience of all emotional states regardless of the valence of the emotion. Thus, RHD patients should have demonstrated a deficit in SCR during the reward as well as the shock condition. In contrast, according to the bivalent view of emotions, the RHD patients were expected to display normal processing of positive emotional experiences, whereas the LHD were expected to show deficiencies in processing of pleasant experiences. Moreover, since SCR did not reliable

distinguish the reward from the reward-control trials, the predictions about group differences based on the global and bivalent models could not be examined.

Yet, when the one LHD subject with high SCRs is removed, the LHD group, as well as the RHD group, appears to be deficient in emotional responding during the shock condition. This finding does not provide support for either the global or the bivalent view of emotional responding.

The verbal report measures showed clear differences in the ratings of subjects during the stimulus compared to the control trials. For both the shock and reward conditions, however, there were no group differences in ratings of emotion. As a consequence, the verbal report data does not support either the global or bivalent models of emotion.

In conclusion, there is a dissociation in RHD patients and most of the LHD subjects between verbal report of emotion and autonomic responding. The reason for this dissociation is unclear. It may be that subjects were able to perceive the emotional situations accurately, but have a deficit in autonomic responding. Another explanation is that the brain damaged subjects were unable to accurately perceive the emotional content of the anticipation trials accurately, and thus, did not exhibit the expected SCR responses. Lastly, a combination of both possibilities may have contributed to the dissociation between verbal report and autonomic responding.

Neuroanatomic Correlates

It is important to examine these findings from the neurobiological perspective. Below some of the current evidence regarding the neural organization of emotion will be reviewed. Based on this information, the current findings will be discussed.

In a recent review of the neurobiology of emotional conditioning, LeDoux (1994) described two pathways responsible for shock conditioning, a cortical and subcortical pathway. LeDoux describe recent work in animals where tones are paired with shock. The tone comes through the ear proceed from the auditory nerves to the auditory midbrain to the auditory thalamus. The auditory thalamus has projections to the primary auditory cortex as well as to the amygdala. In animals, fear conditioning still occurs after bilateral ablation of the primary auditory cortex.

According to LeDoux the cortical system is involved in the slower, top-down interpretation of the emotional significance of the situation. In this study, it remains unclear whether the RHD and most of the LHD accurately interpreted the situation correctly. The lack of SCR findings may be related to the inability of subjects to interpret the anticipatory trials accurately.

Heilman, Watson, and Valenstein (1994), reviewed the literature on reaction time tasks in patients with unilateral lesions which revealed that RHD patients had

slower reaction times regardless of the hand they used in a task. They suggested that because patients with RHD have reduced behavioral evidence of activation, that RHD mediates the activation process. Specifically, these authors suggest that the left hemisphere prepares the right extremities for action, whereas the right hemisphere prepares both sides of the body for responding. Thus, according to this theory, the decreased autonomic responding in the RHD group can be explained by their deficit in global physiological readiness to respond. The decreased SCRs in most of the LHD group can not be explained by this theory.

Tranel and Damasio (1994) examined 36 patients with brain damage who had detailed neuroanatomic evaluations of their lesions. They found two areas in patients with unilateral brain damage which affect SCR to positive and negative emotional slides. One area was the cingulate gyrus in either the right and left hemisphere. The other area was the supramarginal gyrus and angular gyrus on the right side only. In the present study, when subjects were divided into anterior and posterior lesions, right hemisphere patients with posterior lesions had smaller SCRs during the shock condition compared to RHD subjects with anterior lesions. The opposite trend was found in the LHD group. These trends are consistent with the findings of Tranel and Damasio regarding the supramarginal and angular gyri on the right. The differences in responding in patients with lesions

involving the cingulate gyrus could not be examined in the present sample because few patients had lesions involving that area.

Limitations of the Study

There are several limitations of the present study. First, both normal and brain damaged subjects did not show the normal orienting and habituation in the psychophysiological screening. As a consequence, it can not be said that the RHD patients in this study have a specific deficit in electrodermal arousal, as measured by SCR. Tranel and Damasio found that some stroke patients have deficits in orienting and emotional arousal, whereas others have deficits in emotional arousal alone. Other researchers, however, have found patients with RH strokes show normal orienting, but abnormal emotional arousal (Meadows and Kaplan, 1994). The orienting procedure used by Meadows and Kaplan differed from the present study in that they used much louder tone (100 db), whereas the present authors used tones of 60 db. This difference in the intensity of the tones may account for the discrepancy between the current findings and those revealed by Meadows and Kaplan.

Second, none of the psychophysiological measures accurately distinguish the reward from the control situation. Because this suggests that the reward condition was problematic, the global and bivalent models could not be

distinguished using this procedure. Several reasons could account for this problem. As mentioned above, the reward situation is not as emotionally arousing as the shock condition. Since SCR is found to be highly correlated with arousal rating (Greenwald, Cook, and Lang, 1989), the lack of SCR in the reward condition may be related to the lack of arousal experienced by the subjects in this condition. Additionally, facial EMG which have been correlated with ratings of valence, does not appear to be a useful measure in this population.

Third, since subjects were not asked to respond in any way during the anticipatory period, it is unclear whether subjects were accurately interpreting the emotional context of the anticipation period on a trial by trial basis. Although subjects demonstrated competence at distinguishing the high and low pairs of tones from one another before the onset of the experiment, some subjects may have difficulty distinguishing certain tones or remembering the significance of the tones during the experimental procedure. As a consequence, the lack of SCR findings in the RHD group and most of the subjects in the LHD group could possibly be reflective of problems accurately interpreting the significance of the anticipatory period.

Lastly, attempts were made to map each subject's scans onto Damasio's templates. However, this study was not designed to be able to carefully determine the

neuroanatomical areas involved in each lesion. As a consequence, the results of the individual case studies needs to be interpreted with caution.

Future Directions

Attempts need to be made to find more accurate ways to measure pleasant emotional valence. For example, by using younger, female subjects facial EMG may become a useful instrument. There are few young female stroke patients. Perhaps studies using facial EMG would provide more useful results if conducted with different patient populations, i.e., patients with temporal lobectomies.

Another possible way to measure facial expressiveness may be to use a facial coding such as a facial coding system like FACS or the system of digitizing light changes in pixels. FACS has been used successfully in the past as a method to explore facial expressiveness in patients with unilateral brain damage (Mammacuri, et al., 1988; Caltagirone, et al., 1989).

Additionally, perhaps the level of arousal during a reward condition could be raised by providing subjects with immediate money/lottery tickets or possibly increasing the monetary value awarded to the subjects.

Most importantly, if an anticipatory paradigm is used in the future, it will be important to have subjects respond in some way during the anticipation period to insure that they are interpreting each trial accurately. One caveat to

having subjects respond in some way is that their physiological responding, especially heart rate, will be influenced by the motor response.

In sum, in the present study attempts were made to measure multiple emotional response systems by using in vivo emotional experiences in patient with left and right cortical strokes. This study is unique in that pleasant and unpleasant experiences were examined separately, multiple response systems of emotion were measures, and that in vivo rather than perceptual stimuli were used to evoke emotions.

The findings revealed that in normal subjects, skin conductance and verbal report differentiated the shock from the no-shock task. In the reward task, only verbal report clearly differentiated the stimulus from control conditions. Within the shock condition, both RHD and most LHD demonstrated decreased skin conductance responding relative to the normal controls, but had no differences in verbal report. These findings could be reflective of actual deficits in electrodermal arousal or inability to clearly understand the top-down nature of the anticipatory task.

APPENDIX A
PSYCHOLOGICAL MEASURES

Self-Assessment Manikin (SAM)

The Self-Assessment Manikin (SAM) measures subjective ratings of three independent affective dimensions which have been derived from factor analytic studies (Hodes, Cook, & Lang, 1985). The three dimensions include valence (pleasant to unpleasant), arousal (aroused to calm), and control (dominance to submission). There are both computer and paper and pencil versions of SAM. For purposes of this study, a paper and pencil version of SAM in which each dimension is presented as a series of nine cartoon characters will be used. For the valence dimension, SAMs facial expression gradually changes from a smile to a frown. Arousal is denoted by increased activity in the abdomen to no activity and wide eyes to closed eyes. Control is represented from a very large character who gradually shrinks in size to a very small character.

Positive and Negative Affect Schedule

The Positive and Negative Affect Schedule (PANAS) is comprised of two 10-item mood scales. Using factor analysis positive affect (PA) and negative affect (NA) factors were identified. Principle components analysis was employed to

choose the specific descriptors for the schedule. Preliminary analyses revealed that 10 terms were sufficient for each scale. Undergraduate subjects were asked to complete the schedule, reporting their affect for moment, today, past few days, past few weeks, year, and in general. Internal consistency and intercorrelations range from .86 to .90 for PA and .84 to .87 for NA. As expected the correlation between PA and NA is low, ranging from -.12 to -.23. Test-retest reliability after 1 week were .47 to .68 for PA and .39 to .71 for NA. Correlations with Hopkins Symptom Checklist, Beck Depression Inventory, and State-Trait Anxiety Scale (state anxiety) is .51 to .74 for NA and -.19 to .36 for PA.

APPENDIX B
DEMOGRAPHIC INFORMATION

Table B-1 Medications taken by RHD Group

Group	Medications
R1	Digoxin*, Zantac, Nitrobid, Inderal*, Quinine Sulfate, Procardia*
R2	Aspirin, Vitamin B, Docusate, Nifedipine*
R3	Pepcide, Glyburide, Lopressor*, Nalfom
R4	Isosorbate, Atavan*
R6	Dilantin, Aspirin, Lopressor*, Zestril*, Isosorbide, Mevacore
R7	Tegretol, Aspirin, Hydrchlorothizide, Lisinopril*, Glyburide
R8	Lisinopril*, Isordil, B-12, Aspirin
R9	Aspirin, Fosinopril*, Propoxyphene, Quindirine Glucomate, Albuterol Oral
R11	None
R12	Lopid, Vasotec*, Coumadin, Micronase, Chlorzoxazone, Verapamil*, Triazolan, Alprazolan*
R13	Isodil, Aspirin, Lopressor*
R14	Vasotec*, Lopressor*

* affects the autonomic nervous system

Table B-2 Medications taken by the RH NCS

Group	Medications
RC2	Hydrochlorothiazide
RC2	None
RC3	None
RC4	Corgard*, Hytrin*, Diazide
RC5	Tagamet
RC6	Vasotec*
RC7	None
RC8	Clinoril
RC9	Ibprophen, Metiprolol*, Nifedipine*,
RC10	None
RC11	None
RC12	Sudafed

* affects the autonomic nervous system

Table B-3 Medications taken by the LHD Group

Group	Medications
L2	Aspirin, Loniten, Cardura*
L3	Thyroid, Triamcinalone acetonide inhaler, Albuterate, Postassium Chloride, Furosimide, Lisinopril*, Glyburide
L4	None
L5	Aspirin
L6	Digoxin*, Aspirin
L7	Tagemet, Coumadin
L8	Coumadin, Quinidine, Digoxin*, Vasotec*
L9	Atenolol*, Coumadin
L10	Verapamil*, Prednizone, Darvon
L11	Capozide*
L12	Coumadin, Arthritis medication
L13	Aspirin, Lopressor*

Table B-4 Medications taken by LH NCS

Group	Medications
LC1	Glyburide, Metorolol*, Nifidipine*, Monoxidil, Furolcemide, Liscinopril*, Docusate, Cimetidine
LC2	None
LC3	None
LC4	Meclizene, Aspirin
LC5	Arthritis medication
LC6	Aspirin, Loped, Zestril*, Glucontrol
LC7	Micronase, Allopurinol, Aspirin
LC8	Aspirin, Digoxin*, Zocor
LC9	Lopressor*, Aspirin, Lasix
LC10	Voltarian, Calcium, Aspirin
LC11	Mevcor, Zynthryroid
LC12	None

* affects the autonomic nervous system

Table B-5 Neurological Information for the RHD Group

	SEX	AGE	YEARS OF EDUC.	BRODMANN'S AREAS INVOLVED IN CVA	LESION LOCATION	MONTHS SINCE CVA
R8	M	73	18	39, 22, 21, 20, 40, 3,2,1, 6, 4, 46, 9, 11, 47, 45, 44	Mixed	216
R2	M	73	14	6, 7, 40(mixed), 8, SPWM, STWM	Mixed	192
R3	M	74	13	22, 37, 39, 40(mixed), STWM	Posterior	12
R4	M	64	12	3,1,2, 6, 44	Primarily Anterior	14
R6	M	54	8	3,1,2, 19, 21(posterior), 22(posterior), 37, 39, 40(mixed), Medial temporal, SPWM	Posterior	46
R7	M	76	10	4, 6, corona radiata	Mixed	43
R8	M	64	8	3,1,2, 7, 19, 22, 39, 40, 41, 42, insular cortex, medial temporal, STWM	Posterior	62
R9	M	60	8	3,1,2, 6, 9, 10, 24, 25, 32, 44, 45, 46, striatum, internal capsule, corona radiata	Primarily Anterior	8
R11	M	57	13	3,1,2, 6, 22(anterior), 41, 42, insular cortex, striatum	Primarily Anterior	51
R12	M	48	12	Film not available		18
R13	M	65	15	21(mixed), 22(mixed), 37, 38, 44, 45, 40(anterior), striatum, corona radiata	Mixed	15
R14	M	49	22	3,1,2, 6, 21(anterior), 40(anterior), 41, 42, insular cortex, internal capsule, striatum, STWM	Mixed	54

Table B-6 Neurological Information for the LHD Group

	SEX	AGE	YEARS OF EDUC.	BRODMANN'S AREAS INVOLVED IN CVA	LESION LOCATION	MONTHS SINCE CVA
L2	M	72	14	22(posterior), 40(anterior), Insular cortex	Posterior	155
L3	M	76	13	4, 7, 19, 21(posterior), 22(posterior), 37, 39, 40(posterior), 41, 42, insular cortex, SPWM, STWM, medial temporal	Posterior	178
L4	M	67	12	3,1,2, 22(posterior), 41, 42, 39, insular cortex, STWM	Posterior	50
L5	M	60	14	3,1,2, 4, 6, 9, 10, 11, 12, 20, 21, 22, 23, 24, 32, 37, 38, 39, 40, 41, 42, 45, 46, SCWM, thalamus, striatum, medial temporal, insular cortex	Mixed	236
L6	M	68	15	21, 22(mixed), insular cortex	Mixed	58
L7	M	60	15	22(posterior), 37, 40 (mixed)	Posterior	62
L8	M	72	12	22(posterior), 39, 40(mixed), STWM	Posterior	33
L9	M	60	14	6, 8, 24, corona radiata	Anterior	5
L10	M	68	8	17, 18, 19, 31, medial temporal	Posterior	7
L11	F	76	8	312, 6, 41, 42, 44, 45, insular cortex, striatum, internal capsule	Primarily Anterior	26
L12	M	70	12	3,1,2, 19, 22(posterior), 39, 40(mixed), insular cortex, striatum, medial temporal	Primarily Posterior	51
L13	M	62	16	3,1,2, 6, striatum, internal capsule, STWM, insular cortex	Mixed	75

Table B-7 Means and Standard Deviations on Neuropsychological Testing by Group

Tests	LHD	LH NCS	RHD	RH NCS
WAIS-R, Information	7.25 (3.33)	10.58 (3.94)	9.50 (3.00)	11.00 (3.28)
WAIS-R, Similarities	7.00 (3.16)	9.42 (2.94)	7.83 (3.01)	8.83 (2.95)
Digit Span, Forward	5.25 (1.66)	6.25 (.62)	5.83 (1.12)	8.83 (2.95)
Digit Span, Backwards	3.17 (1.47)	4.33 (1.07)	3.83 (1.27)	5.17 (1.27)
WMS-R Orientation	13.45 (.82)	13.92 (.29)	13.08 (1.65)	13.92 (.29)
WMS-R, Logical Memory I	41.92 (34.26)	65.58 (27.44)	49.08 (32.42)	63.00 (27.09)
WMS-R, Logical Memory II	46.75 (32.44)	67.25 (28.44)	36.83 (27.66)	65.75 (27.01)
WMS-R, Visual Reproduction I	61.33 (31.29)	77.25 (24.31)	42.67 (33.65)	76.67 (30.42)
WMS-R, Visual Reproduction II	52.08 (30.14)	76.17 (25.91)	35.00 (35.72)	71.17 (36.36)
WAB, Comprehension	9.22 (.74)	9.73 (.39)	9.73 (.39)	9.85 (.21)
WAB, Aphasia Quiotent	92.00 (8.17)	98.67 (1.07)	97.87 (2.18)	98.34 (1.31)

Note: WAIS-R scores are presented as standard scores, digit span scores are presented as # of digits, WMS-R orientation scores are reported as raw score with a high score of 14, WMS-R Logical Memory and Visual Reproduction scores are presented as percentiles based on age-related norms, WAB comprehension is the # correct out of 10, WAB aphasia quoitent is the # correct out of 100.

Table B-8 Results of Neuropsychological Testing for RHD Patients

ID	WAIS-R INFO	WAIS-R SIM	DIGIT SPAN	WMS-R Orient	WMS-R LOG MEM I & II	WMS-R VIS REPRO I & II	NEGLECT	FACIAL RECOGN	APHASIA SCORE
R1	SS=12	SS=11	4/4	13	88/42	34/26	No	SEVERE	10/99.4
R2	SS=11	SS=5	7/5	13	21/18	8/10	Neg, Ext	SEVERE	9.7/96.2
R3	SS=10	SS=5	5/5	14	5/4	34/10	Neg, Ext	SEVERE	9.65/96.7
R4	SS=16	SS=11	6/4	14	53/53	54/73	No	BORDERLINE	10/100
R6	SS=10	SS=9	4/2	12	29/19	15/23	Ext	BORDERLINE	10/95.8
R7	SS=5	SS=4	7/2	11	74/46	20/6	No	WNL	9.1/93.4
R8	SS=10	SS=7	5/4	14	11/15	19/11	Neg, Ext	SEVERE	9.95/98.9
R9	SS=8	SS=7	6/2	11	80/64	2/1	Neg, Ext	SEVERE	8.85/95.9
R11	SS=6	SS=6	7/4	14	90/92	95/59	No	WNL	10/100
R12	SS=6	SS=9	6/4	13	9/10	50/10	?Neg, Ext	SEVERE	10/99.4
R13	SS=10	SS=6	7/4	14	72/65	83/92	No	BORDERLINE	10/100
R14	SS=10	SS=14	6/6	14	57/14	98/99	No	WNL	9.55/98.7

Table B-9 Results of Neuropsychological Testing for RH NC Group

ID	WAIS-R INFO	WAIS-R SIM	DIGIT SPAN	WMS-R Orient	WMS-R LOG MEM I & II	WMS-R VIS REPRO I & II	NEGLECT	FACIAL RECOGN	APHASIA SCORE
RC1	SS=9	SS=6	6/4	14	80/78	98/98	No	WNL	10/98
RC2	SS=15	SS=16	8/7	14	94/97	99/99	No	WNL	10/99.8
RC3	SS=11	SS=9	5/5	14	12/26	99/99	No	WNL	9.9/97.6
RC4	SS=15	SS=11	7/5	14	76/42	98/98	No	WNL	10/99.6
RC5	SS=8	SS=8	6/3	14	72/81	89/93	No	WNL	9.6/99.2
RC6	SS=13	SS=8	8/7	14	68/72	54/66	No	WNL	10/98.6
RC7	SS=10	SS=5	8/5	14	27/48	9/7	No	MODERATE	9.35/95.7
RC8	SS=12	SS=8	7/5	14	90/98	98/96	No	WNL	10/98.4
RC9	SS=15	SS=12	7/7	14	83/97	77/77	No	WNL	10/99.6
RC10	SS=6	SS=7	7/5	13	76/76	33/13	No	WNL	9.7/96.2
RC11	SS=6	SS=8	6/4	14	49/51	68/88	No	WNL	9.75/98.3
RC12	SS=12	SS=8	7/5	14	29/23	32/20	No	WNL	9.85/99.1

Table B-10 Results of Neuropsychological Testing for LHD Group

	WAIS-R INFO	WAIS-R SIM	DIGIT SPAN	WMS-R Orient	WMS-R LOG MEM I & II	WMS-R VIS REPRO I & II	NEGLECT	FACIAL RECOGN	APHASIA SCORE
L2	SS=10	SS=7	8/6	12	98/97	72/68	No	SEVERE	9.5/98
L3	SS=7	SS=7	3/0	14	6/8	60/46	Ext	WNL	8.2/86
L4	SS=6	SS=6	4/3	14	54/40	76/21	No	WNL	9.3/88.8
L5	SS=1	SS=2	4/3	NA	2/5	66/	Ext	MODERATE	7.65/69.5
L6	SS=7	SS=10	5/4	14	52/47	98/93	No	WNL	9.4/96.6
L7	SS=12	SS=13	7/4	14	59/85	88/80	No	WNL	10/100
L8	SS=10	SS=9	5/2	14	6/26	76/90	No	WNL	8.7/89.2
L9	SS=12	SS=10	8/4	14	98/92	66/46	No	WNL	9.5/94.2
L10	SS=4	SS=3	6/3	13	11/15	2/1	No	WNL	8.8/94.8
L11	SS=4	SS=4	5/3	13	17/26	2/8	No	MODERATE	9.95/95.5
L12	SS=7	SS=7	4/2	12	60/56	42/64	No	WNL	9.65/95.5
L13	SS=7	SS=6	4/4	14	40/64	88/42	No	WNL	9.95/95.9

Table B-11 Results of Neuropsychological Testing for LH NC Group

ID	WAIS-R INFO	WAIS-R SIM	DIGIT SPAN	WMS-R Orient	WMS-R LOG MEM I & II	WMS-R VIS REPRO I & II	NEGLECT	FACIAL RECOGN	APHASIA SCORE
LC1	SS=6	SS=5	5/3	14	67/68	68/59	No	WNL	
LC2	SS=7	SS=8	6/4	14	94/97	83/82	No	WNL	10/99.4
LC3	SS=13	SS=12	6/4	13	57/81	94/99	No	BORDERLINE	10/100
LC4	SS=6	SS=6	7/3	14	21/36	94/84	No	WNL	9.8/97.8
LC5	SS=13	SS=15	6/4	14	40/18	64/64	No	WNL	9.95/98.1
LC6	SS=14	SS=8	7/4	14	23/32	52/94	No	WNL	10/97.6
LC7	SS=14	SS=9	6/5	14	89/87	99/99	No	WNL	10/100
LC8	SS=14	SS=7	6/6	14	78/76	99/98	No	WNL	9.85/98.5
LC9	SS=13	SS=13	6/3	14	98/98	99/98	No	WNL	9.85/98.9
LC10	SS=15	SS=11	6/5	14	98/98	99/67	No	WNL	10/100
LC11	SS=7	SS=10	7/6	14	68/76	42/58	No	WNL	9.95/96.9
LC12	SS=5	SS=9	7/5	14	54/40	34/12	No	WNL	10/98.2

Table B-12 Performance of RHD on Florida Affect Battery (percent correct)

ID	1	2	3	4	5	6	7	8a	8b	9
R1	75	65	85	90	50	50	85	60	50/13	30
R2	50	65	80	80	75	88	80	75	75/31	65
R3	75	65	55	90	60	75	65	40	94/25	20
R4	90	80	60	100	80	75	95	75	100/63	80
R6	80	90	75	95	65	100	95	90	75/69	80
R7	85	70	75	75	50	33	90	85	50/44	55
R8	75	80	65	80	60	100	100	75	81/50	60
R9	55	60	60	75	55	50	70	70	88/13	45
R11	95	65	100	95	50	88	95	85	100/44	85
R12	90	80	90	90	75	100	95	75	75/69	90
R13	75	80	75	95	70	69	70	55	100/60	70
R14	100	85	90	100	85	100	85	100	81/75	100

Table B-12 Performance of RHD on Florida Affect Battery (percent correct)

ID	1	2	3	4	5	6	7	8a	8b	9
R1	75	85	85	80	80	50	80	60	50/13	40
R2	80	65	80	80	75	88	80	75	75/31	65
R3	85	65	55	90	60	75	65	44	94/25	20
R4	85	80	90	100	80	75	95	75	100/63	80
R5	80	90	75	85	60	100	95	90	75/69	80
R7	85	70	75	75	50	44	90	25	50/44	55
R8	75	80	65	80	60	100	100	75	81/50	60
R9	55	90	60	75	55	50	70	70	88/13	45
R11	95	65	100	85	80	88	95	85	100/44	45
R12	90	80	90	90	75	100	95	75	75/69	90
R13	75	50	75	95	70	69	70	55	100/60	70
R14	100	85	90	100	85	100	85	100	81/75	100

Table B-13 Performance of RH NCS on Florida Affect Battery (percent correct by subtest)

ID	1	2	3	4	5	6	7	8a	8b	9
RC1	100	95	85	85	95	100	90	85	94/ 50	60
RC2	100	85	90	100	95	94	100	100	88/ 44	100
RC3	90	90	75	95	85	88	90	75	75/ 69	75
RC4	95	85	100	100	95	100	100	100	100 /69	100
RC5	100	90	75	95	85	100	100	95	94/ 63	60
RC6	95	85	75	100	85	100	100	90	94/ 75	95
RC7	100	85	75	85	55	75	95	65	88/ 50	60
RC8	100	90	95	95	90	100	100	85	94/ 69	95
RC9	90	85	85	100	95	100	100	100	81/ 75	90
RC10	100	80	65	100	85	94	90	55	88/ 44	60
RC11	95	80	90	100	80	100	100	95	88/ 69	90
RC12	100	65	80	90	95	100	100	95	81/ 81	80

Table B-14 Performance of LHD on Florida Affect Battery (percent correct by subtest)

ID	1	2	3	4	5	6	7	8a	8b	9
L2	100	90	90	95	90	100	100	85	81/63	80
L3	90	85	95	85	90	69	90	75	94/63	65
L4	100	70	35	100	80	94	100	80	75/63	80
L5	90	80	95	100	100	94	50	75	63/44	75
L6	100	90	85	100	90	100	100	80	94/75	95
L7	100	80	90	95	95	100	100	80	81/75	90
L8	100	75	90	100	75	100	100	75	81/56	90
L9	95	85	90	100	80	100	100	100	87/75	85
L10	90	75	85	90	70	69	95	60	81/13	65
L11	70	60	70	90	85	88	95	90	94/25	60
L12	80	70	75	90	75	100	100	80	94/50	70
L13	85	70	85	95	80	88	90	90	94/56	90

Table B-15 Performance of LH NCS on Florida Affect Battery (percent correct by subtest)

ID	1	2	3	4	5	6	7	8a	8b	9
LC1	100	85	90	95	75	69	100	75	75/50	80
LC2	90	90	90	100	95	94	100	100	100/81	100
LC3	95	85	90	90	90	100	95	100	88/94	95
LC4	100	85	75	100	90	100	100	70	94/38	75
LC5	90	85	85	90	90	100	70	70	81/44	90
LC6	100	85	85	100	100	100	100	90	100/75	90
LC7	100	75	100	100	90	100	100	70	94/50	75
LC8	90	90	90	100	80	100	85	70	81/56	60
LC9	50	90	80	95	90	88	100	70	88/50	90
LC10	85	85	90	95	100	100	100	100	88/94	95
LC11	90	90	75	80	50	94	100	95	94/69	95
LC12	95	85	90	95	80	100	100	70	81/50	80

APPENDIX C
STATISTICAL INFORMATION

Table C-1 ANOVA Table of Mean Heart Rate Change during Psychophysiological Screening

	SS	DF	MS	F	Sig of F
Group	2.228	3	.743	.419	.7404
Subject(Group)	76.260	43	1.773		
Tone	2.814	1	2.814	1.634	.2080
Tone by Group	1.125	3	.375	.218	.8835
Tone by Subject(Group)	74.059	43	1.722		

Table C-2 ANOVA Table of D1 during Psychophysiological Screening

	SS	DF	MS	F	SIG of F
Group	258.12	3	86.04	1.73	.1760
Subject(Group)	2144.46	43	49.87		
Tone	161.89	1	161.89	8.63	.0053
Tone by Group	30.87	3	10.29	.5482	.6521
Tone by Subject(Group)	807.04	43	18.77		
Block	121.30	7	17.33	1.3985	.2054
Block by Group	176.37	21	8.40	.6778	.8544
Block by Subject(Group)	3729.46	301	12.39		
Tone by Block	142.15	7	20.31	1.227	.2875
Tone by Block by Group	330.67	21	15.74	.9514	.5246
Tone by Block by Subject(Group)	4981.73	301	16.55		

Table C-3 ANOVA Table of Percentage of SCR Responses during Psychophysiological Screening

	SS	DF	MS	F	Sig of F
Group	9348.16	3	3116.05	2.037	.1229
Subject(Group)	65787.31	43	15299.93		
Tone	14.76	1	14.76	.200	.6570
Tone by Group	53.55	3	17.85	.242	.8667
Tone by Subject(Group)	3173.68	43	73.81		

Table C-4 ANOVA Table of Recoded Range Corrected SCR during Psychophysiological Screening

	SS	DF	MS	F	SIG OF F
Group	19797.76	3	6599.3	1.91	.1421
Subject(Group)	148487.88	43	3453.2		
Block	1880.28	7	268.61	1.20	.3017
Block by Group	3297.22	21	157.01	.702	.8305
Block by Subject(Group)	67297.82	301	223.58		
Tone	69.80	1	69.80	.209	.6495
Tone by Group	328.28	3	109.43	.328	.8049
Tone by Subject(Group)	14330.56	43	333.27		
Block by Tone	1117.39	7	159.63		.7574
Block by Tone by Group	4902.51	21	233.45	.598	.6244
Block by Tone by Subject(Group)	80333.97	301	266.89	.875	

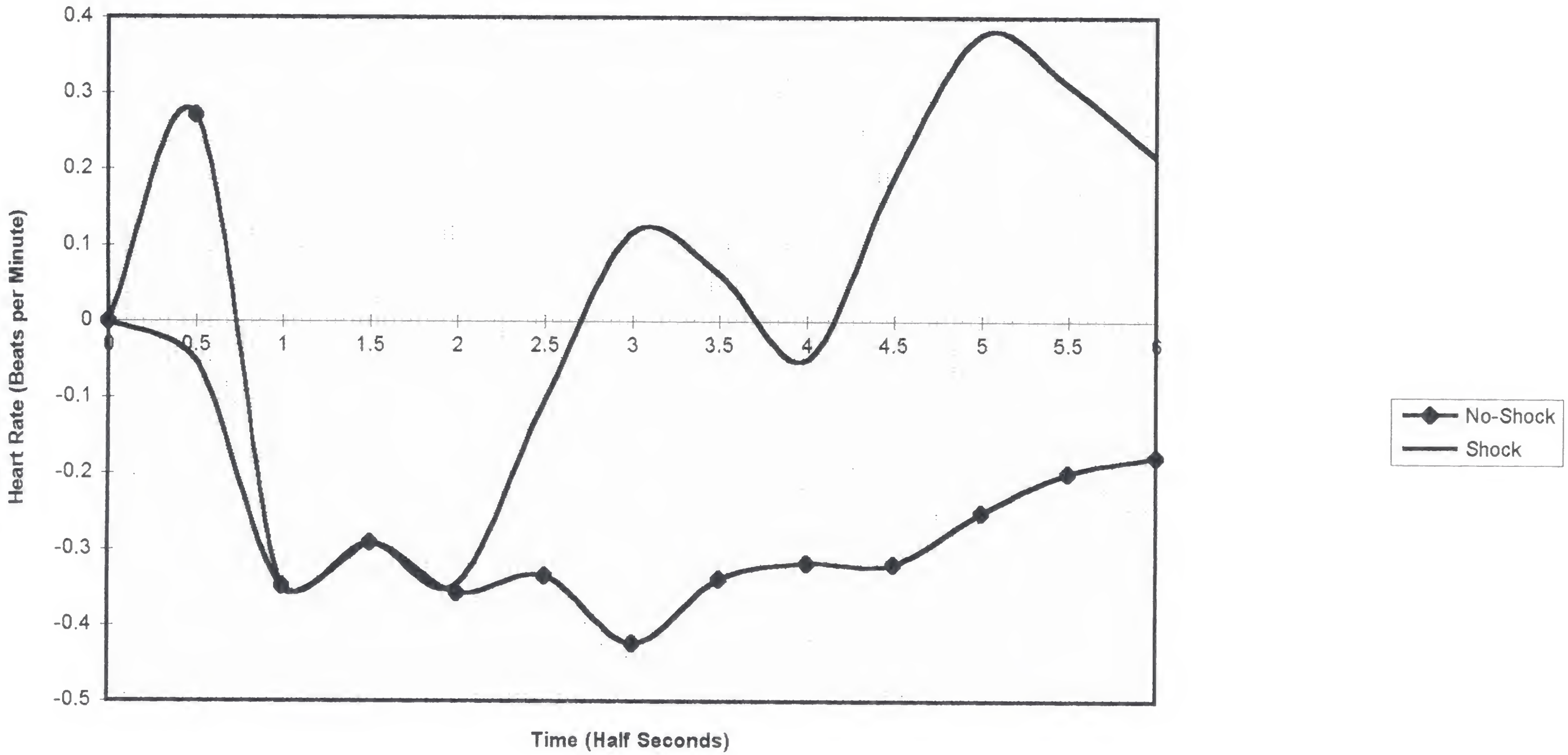

Figure C-1 Heart Rate Change Scores in NCs during Shock Task

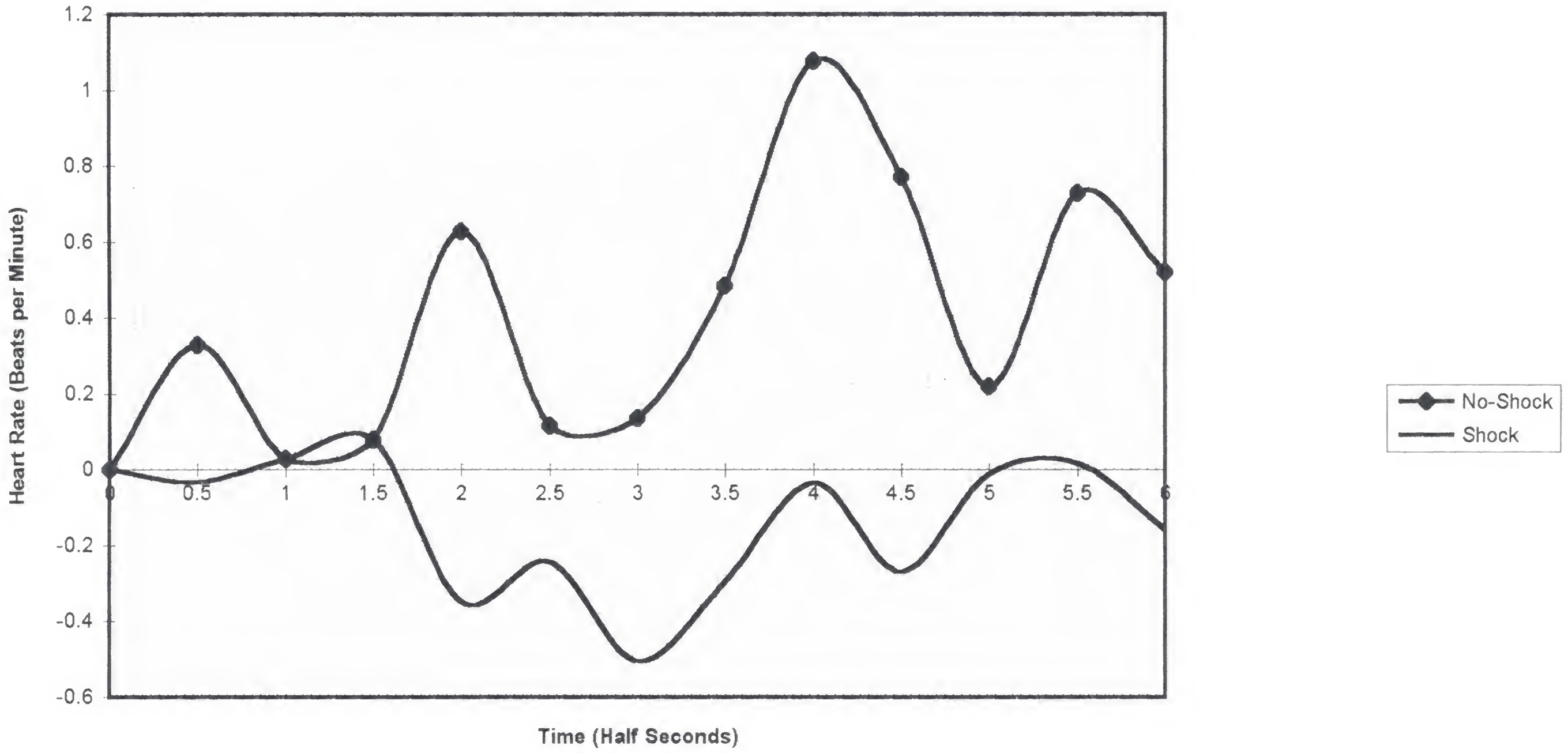

Figure C-2 Heart Rate Change Scores in RHD Ss during Shock Task

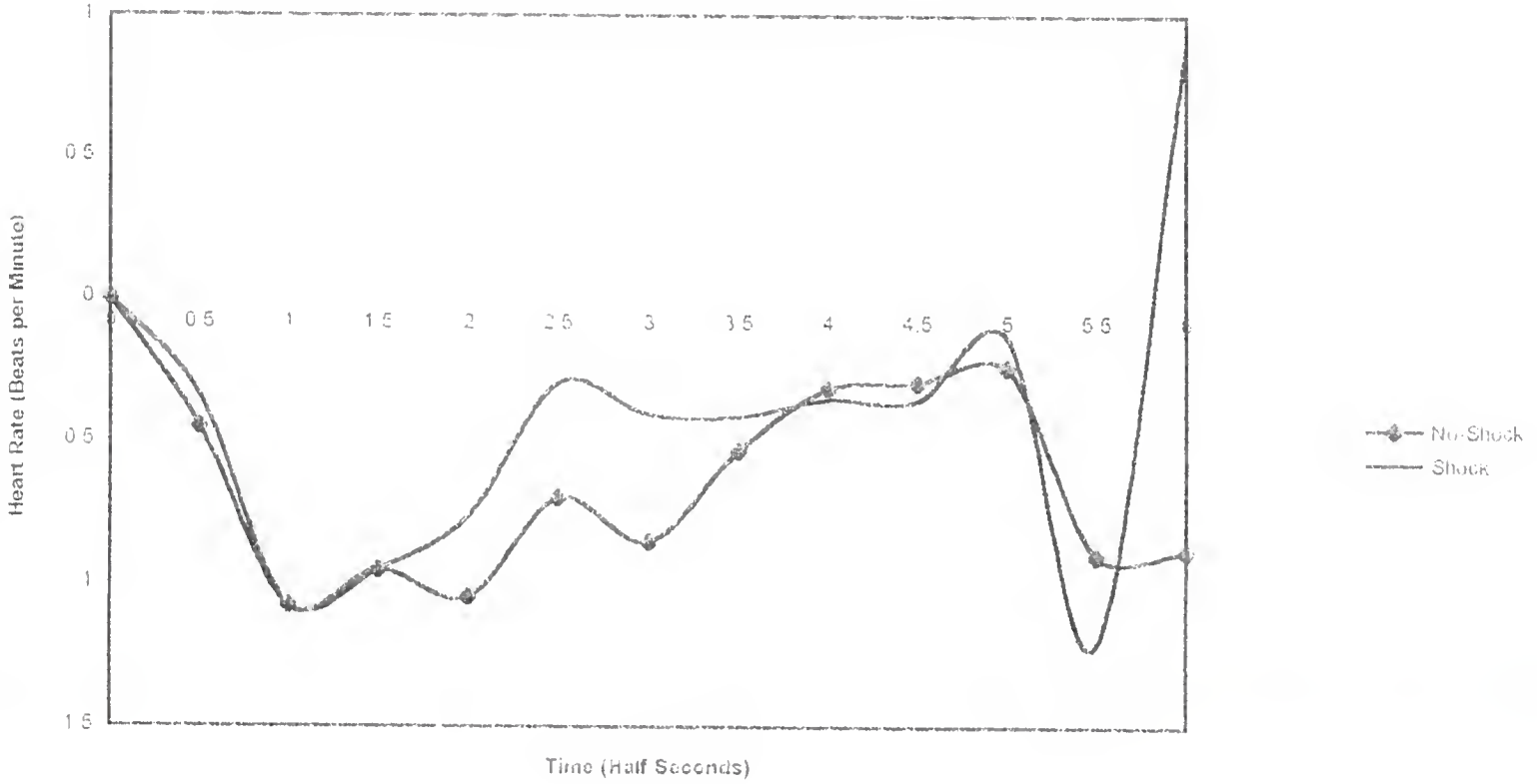

Figure C-3 Heart Rate Change Scores in LHD Ss during Shock Task

Table C-5 ANOVA Table of Mean HR Change from baseline during Shock Task

	SS	DF	MS	F	SIG of F
Group	6.001	3	2.000	1.557	.2137
Subject(Group)	55.246	43	1.285		
Tone	.061	1	.061	.050	.8236
Tone by Group	3.347	3	1.116	.927	.4361
Tone by Subject (Group)	51.766	43	1.204		

Table C-6 ANOVA Table of D1 during Shock Task

	SS	DF	MS	F	SIG of F
Group	51.64414	3	17.2147 1	2.738	.0550
Subject (Group)	270.31	43	6.28629		
Tone	1.236	1	1.23648	.20271	.6548
Tone by Group	7.53	3	2.51059	.41159	.7455
Tone by Subject (Group)	262.288	43	6.09973		
Block	1.85	3	.618	.18456	.9067
Block by Group	44.505	9	4.945	1.4761	.1634
Block by Subject (Group)	432.15	129	3.350		
Tone by Block	12.01	3	4.004	.84401	.4722
Tone by Block by Group	89.38	9	9.931	2.0930	.0346
Tone by Block by Subject(Group)	612.09	129	4.745		

Table C-7 ANOVA Table of D1 during the Shock Condition of the Shock Task

	SS	DF	MS	F	Sig of F
Group	10.978	3	3.659	.4798	.6980
Subject(Group)	327.952	43	7.627		
Block	4.743	3	1.581	.3504	.7889
Block by Group	63.939	9	7.104	1.574	.1295
Block by Subject(Group)	582.118	129	4.512		

Table C-8 ANOVA Table of D1 during the No-Shock Condition of the Shock Task

	SS	DF	MS	F	Sig of F
Group	48.198	3	16.066	3.376	.0268
Subject(Group)	204.647	43	4.759		
Block	9.126	3	3.042	.849	.4695
Block by Group	69.946	9	7.772	2.169	.0282
Block by Subject(Group)	462.121	129	3.582		

Table C-9 T-tests of Group Differences in D1 during the No-Shock Condition of the Shock Task

	Mean Diff.	DF	T-Value	P-Value
LHD, CONS	-.748	33	-1.802	.0807
LHD, RHD	-1.144	22	-2.605	.0162
RHD, CONS	-.396	33	-1.015	.3174

Table C-10 T-Tests of D1 during the No-Shock Condition of Block 1 during the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	.985	33	1.353	.1852
LHD, RHD	.425	22	.633	.5331
RHD, CONS	-.560	33	-.860	.3962

Table C-11 T-Tests of D1 during the No-Shock Condition of Block 2 during the Shock Task

	Mean. Diff.	DF	T-value	P-value
LHD, CONS	-1.773	33	-2.103	.0432
LHD, RHD	-2.183	22	-2.624	.0155
RHD, CONS	-.410	33	-.596	.5555

Table C-12 T-Tests of D1 during the No-Shock Condition of Block 3 during the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-2.063	33	-2.515	.0170
LHD, RHD	-1.717	22	-1.579	.1285
RHD, CONS	.346	33	.546	.5890

Table C-13 T-Tests of D1 duirng the No-Shock Condition of Block 4 during the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-.140	33	-.240	.8117
LHD, RHD	-1.100	22	-1.453	.1604
RHD, CONS	-.960	33	-1.556	.1293

Table C-14 ANOVA Table of A1 during the Shock Task

	SS	DF	MS	F	SIG of F
Group	43.615	3	14.538	.5841	.6287
Subject(Group)	1070.271	43	24.890		
Tone	7.662	1	7.662	.5711	.4539
Tone by Group	80.463	3	26.821	1.999	.1283
Tone by Subject(Group)	576.846	43	13.415		
Block	5.838	3	1.946	.2105	.8890
Block by Group	93.773	3	10.419	1.127	.3483
Block by Subject(Group)	1192.472	129	9.244		
Tone by Block	12.345	3	4.115	.4860	.6926
Tone by Block by Group	101.138	9	11.238	1.3273	.2290
Tone by Block by Subject(Group)	1092.172	129	8.466		

Table C-15 ANOVA Table of D2 during the Shock Task

	SS	DF	MS	F	SIG of F
Group	19.510	3	6.503	1.129	.3479
Subject(Group)	247.622	43	5.759		
Tone	6.757	1	6.757	.835	.3660
Tone by Group	27.798	3	9.266	1.144	.3419
Tone by Subject(Group)	348.099	43	8.095		
Block	6.425	3	2.142	.563	.6401
Block by Group	80.236	9	8.915	2.345	.0175
Block by Subject(Group)	490.355	129	3.801		
Tone by Block	10.192	3	3,397	.644	.5879
Tone by Block by Group	78.850	9	8.761	1.662	.1047
Tone by Block by Subject(Group)	680.128	129	5.272		

Table C-16 ANOVA Table of Block 1 of D2 during the Shock Task

	SS	DF	MS	F	Sig of F
Group	7.390	3	2.463	.9224	.4381
Residual	114.833	43	2.671		

Table C-17 ANOVA Table of Block 2 of D2 during the Shock Task

	SS	DF	MS	F	Sig of F
Group	32.772	3	10.924	5.557	.0026
Residual	84.522	43	1.966		

Table C-18 ANOVA Table of Block 3 of D2 during the Shock Task

	SS	DF	MS	F	Sig of F
Group	4.335	3	1.445	.7053	.5541
Residual	88.092	43	2.049		

Table C-19 ANOVA Table of Block 4 of D2 during the Shock Task

	SS	DF	MS	F	Sig of F
Group	5.377	3	1.792	.9452	.4272
Residual	81.541	43	1.896		

Table C-20 T-Tests of Group Differences in D2 during Block 2 of the Shock Task

Group	Mean Diff.	DF	T-Value	P-Value
LHD, CONS	-1.582	33	-3.263	.0026
LHD, RHD	-2.188	22	-3.273	.0035
RHD, CONS	-.605	33	-1.362	.1824

Table C-21 ANOVA Table of Percentage of SCR Responses during the Shock Task

	SS	DF	MS	F	SIG of F
Group	12897.72	3	4299.242	3.313	.0287
Subject(Group)	55802.27	43	1297.727		
Trial	2481.894	1	2481.894	29.524	.0001
Trial by Group	1630.972	3	543.657	6.467	.0010
Trial by Subject(Group)	3614.773	43	84.064		

Table C-22 T-Tests of Percentage of SCR during the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-19.167	34	-1.944	.0602
LHD, RHD	2.652	21	.290	.7744
RHD, CONS	21.818	33	2.304	.0276

Table C-23 T-Tests of Percentage of SCR Response during the Shock Condtion of the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-27.917	34	-2.582	.0143
LHD, RHD	1.250	21	.143	.8873
RHD, CONS	29.167	33	2.734	.0100

Table C-24 T-Tests of Percentage of SCR Response during the No-Shock Condition of the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-10.417	34	-1.095	.2811
LHD, RHD	4.053	21	.416	.6815
RHD, CONS	14.470	33	-1.627	.1133

Table C-25 ANOVA Table of Recoded Range Corrected SCR during the Shock Task

	SS	DF	MS	F	SIG of F
Group	7403.641	3	2467.880	2.989	.0414
Subject(Group)	35502.724	43	825.645		
Block	3987.684	3	1329.228	14.059	.0001
Block by Group	1123.810	3	124.868	1.321	.2323
Block by Subject(Group)	12196.852	129	94.549		
Tone	4191.208	1	4191.208	23.357	.0001
Tone by Group	3551.953	3	1183.984	6.5980	.0009
Tone by Subject(Group)	7716.130	43	179.445		
Block by Tone	934.601	3	311.534	3.829	.0115
Block by Tone by Group	355.217	9	39.469	.485	.8825
Block by Tone by Subject(Group)	10495.213	129	81.358		

Table C-26 T-Tests of Group Differences in Recoded Range Corrected SCR during the Shock Task

Group	Mean Diff.	DF	T-Value	P-Value
LHD, CONS	-6.179	34	-1.544	.1319
LHD, RHD	3.036	21	.792	.4373
RHD, CONS	9.215	33	2.601	.0138

Table C-27 T-Tests of Block Differences in Recoded Range Corrected SCR during the Shock Task

Block	Mean Diff.	DF	T-Value	P-Value
Block 1, Block 2	6.290	46	4.244	.0001
Block 1, Block 3	8.044	46	4.572	<.0001
Block 1, Block 4	7.910	46	4.303	<.0001
Block 2, Block 3	1.754	46	1.812	.0765
Block 2, Block 4	1.620	46	1.396	.1694
Block 3, Block 4	-.134	46	-.115	.9092

Table C-28 T-Tests of Condition Differences in Recoded Range Corrected SCR by Block during the Shock Task

Blocks	Mean Diff.	DF	T-Value	P-Value
Block 1	-11.345	46	-4.655	<.0001
Block 2	-4.751	46	-2.264	.0284
Block 3	-3.089	46	-1.449	.1542
Block 4	-8.101	46	-3.433	.0013

Table C-29 T-Tests of Recoded Range Corrected SCR during the No-Shock Condition of the Shock Task

	Mean Diff	DF	T	P
LHD, CONS	-.726	34	-.417	.6792
LHD, RHD	.054	21	.029	.9775
RHD, CONS	.780	33	.4815	.6335

Table C-30 T-Tests of Recoded Range Corrected SCR during the NO-No-Shock Condition of the Shock Task

	Mean Diff	DF	T	P
LHD, CONS	-11.529	34	-2.152	.0386
LHD, RHD	3.840	21	.919	.3685
RHD, CONS	-15.834	33	3.099	.0040

Table C-31 ANOVA Table of Corrugator EMG during the Shock Task

	SS	DF	MS	F	SIG of F
Group	.038	3	.013	.287	.8349
Subject(Group)	1.948	44	.044		
Block	.004	3	.001	.127	.9438
Block by Group	.032	9	.003	.356	.9536
Block by Subject(Group)	1.31	132	.010		
Tone	.013	1	.013	1.246	.2703
Tone by Group	.015	3	.005	.467	.7071
Tone by Subject(Group)	.457	44	.010		
Block by Tone	.020	3	.007	.627	.5989
Block by Tone by Group	.110	9	.01●	1.157	.3276
Block by Tone by Subject(Group)	1.390	132	.011		

Table C-32 ANOVA Table of Right-sided Zygomatic EMG during the Shock Task

	SS	DF	MS	F	SIG of F
Group	.026	3	.009	.365	.7788
Subject(Group)	1.038	44	.023		
Block	.023	3	.008	1.148	.3321
Block by Group	.097	9	.011	1.641	.1100
Block by Subject(Group)	.866	132	.007		
Tone	.007	1	.007	.952	.3346
Tone by Group	.051	3	.017	2.35	.0856
Tone by Subject(Group)	.316	44	.007		
Block by Tone	.032	3	.011	.982	.4035
Block by Tone by Group	.101	9	.011	1.035	.4157
Block by Tone by Subject(Group)	1.438	132	.011		

Table C-33 ANOVA Table of Left-sided Zygomatic during the Shock Task

	SS	DF	MS	F	SIG of F
Group	.054	3	.018	1.316	.2811
Subject(Group)	.605	44	.010		
Block	.010	3	.003	.446	.7203
Block by Group	.158	3	.018	2.451	.0130
Block by Subject(Group)	.945	132	.007		
Tone	.010	1	.010	1.417	.2403
Tone by Group	.032	3	.010	1.449	.2416
Tone by Subject(Group)	.323	44	.007		
Block by Tone	.007	3	.002	.171	.9157
Block by Tone by Group	.070	9	.008	.605	.7913
Block by Tone by Subject(Group)	1.700	132	.013		

Table C-34 ANOVA Table of Left-sided Zygomatic EMG for Block 1 of the Shock Task

	SS	DF	MS	F	Sig of F
Group	.041	3	.0136	3.054	.0382
Residual	.195	44	.0044		

Table C-35 ANOVA Table of Left-sided Zygomatic EMG for Block 2 of the Shock Task

	SS	DF	MS	F	Sig of F
Group	.0303	3	.0101	1.657	.1901
Residual	.2684	44	.0061		

Table C-36 ANOVA Table of Left-sided Zygomatic EMG for Block 3 of the Shock Task

	SS	DF	MS	F	Sig of F
Group	.0144	3	.0048	1.543	.2167
Residual	.1367	44	.0031		

Table C-37 ANOVA Table of Left-sided Zygomatic EMG for Block 4 of the Shock Task

	SS	DF	MS	F	Sig of F
Group	.0208	3	.0048	1.743	.1720
Residual	.1748	44	.0040		

Table C-38 T-Tests of Group Differences in Block 1 of Left-sided Zygomatic EMG during the Shock Task

Group	Mean Diff.	DF	T-Value	P-Value
LHD, LH NCS	-.060	22	-2.264	.0338
LHD, RHD	-.067	22	-2.091	.0483
LHD, RH NCS	-.072	22	-2.451	.0227
LH NCS, RHD	-.007	22	-.288	.7763
LH NCS, RH NCS	-.012	22	-.557	.5829
RHD, RH NCS	-.004	22	-.164	.8713

Table C-39 Kruskal-Wallis Tests of SAM Ratings during Shock Task

			Corrected for Ties	
	Chi-Square	Significance	Chi-Square	Significance
Valence (Shock)	1.993	.5734	2.0433	.5635
Valence (Control)	.0410	.9978	.0612	.9960
Arousal (Shock)	1.2324	.7453	1.2654	.7374
Arousal (Control)	1.2959	.7301	1.8491	.6043
Dominance (Shock)	1.8565	.6027	2.0384	.5645
Dominance (Control)	.2111	.9758	.3150	.9572

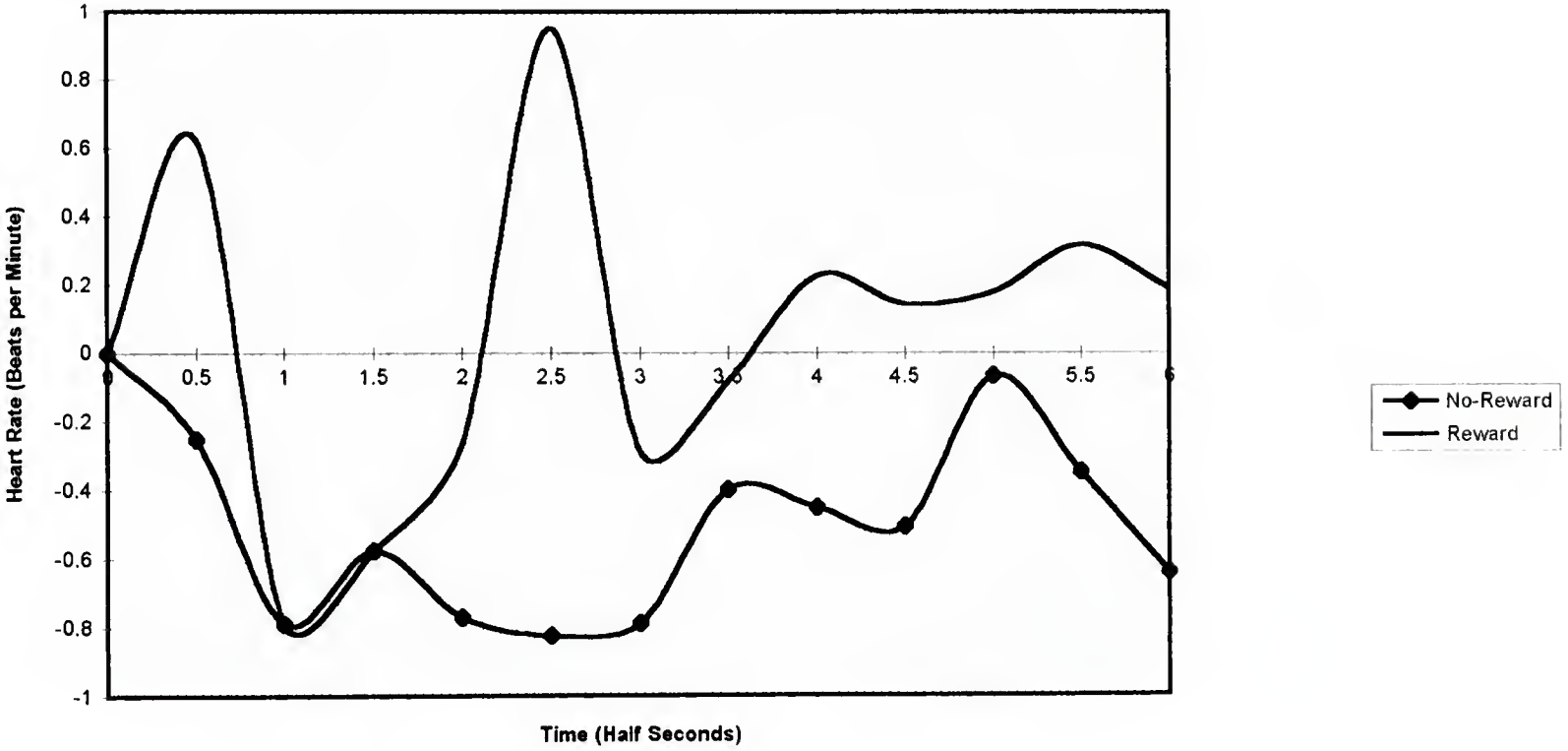

Figure C-4 Heart Rate Change Scores in NCs during Reward Task

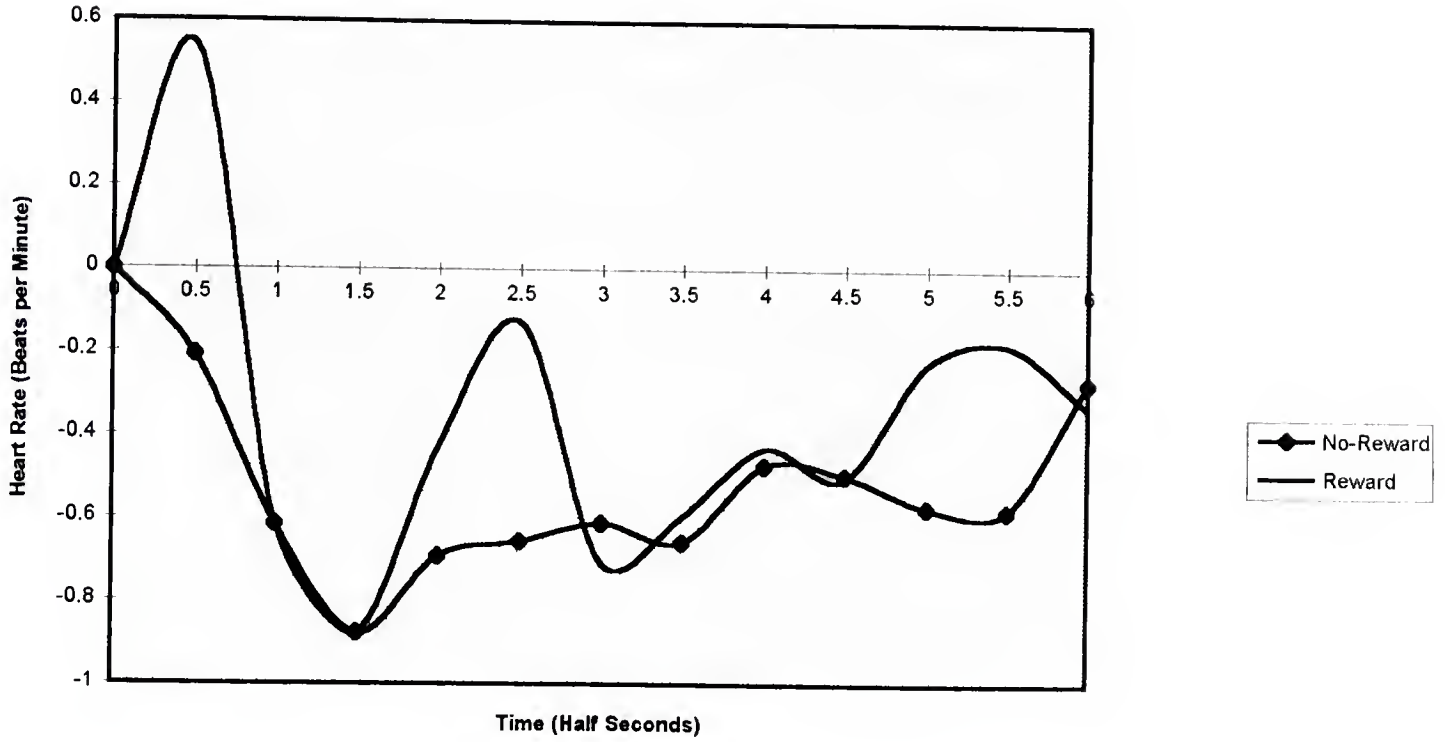

Figure C-5 Heart Rate Change Scores in RHD Ss during Reward Task

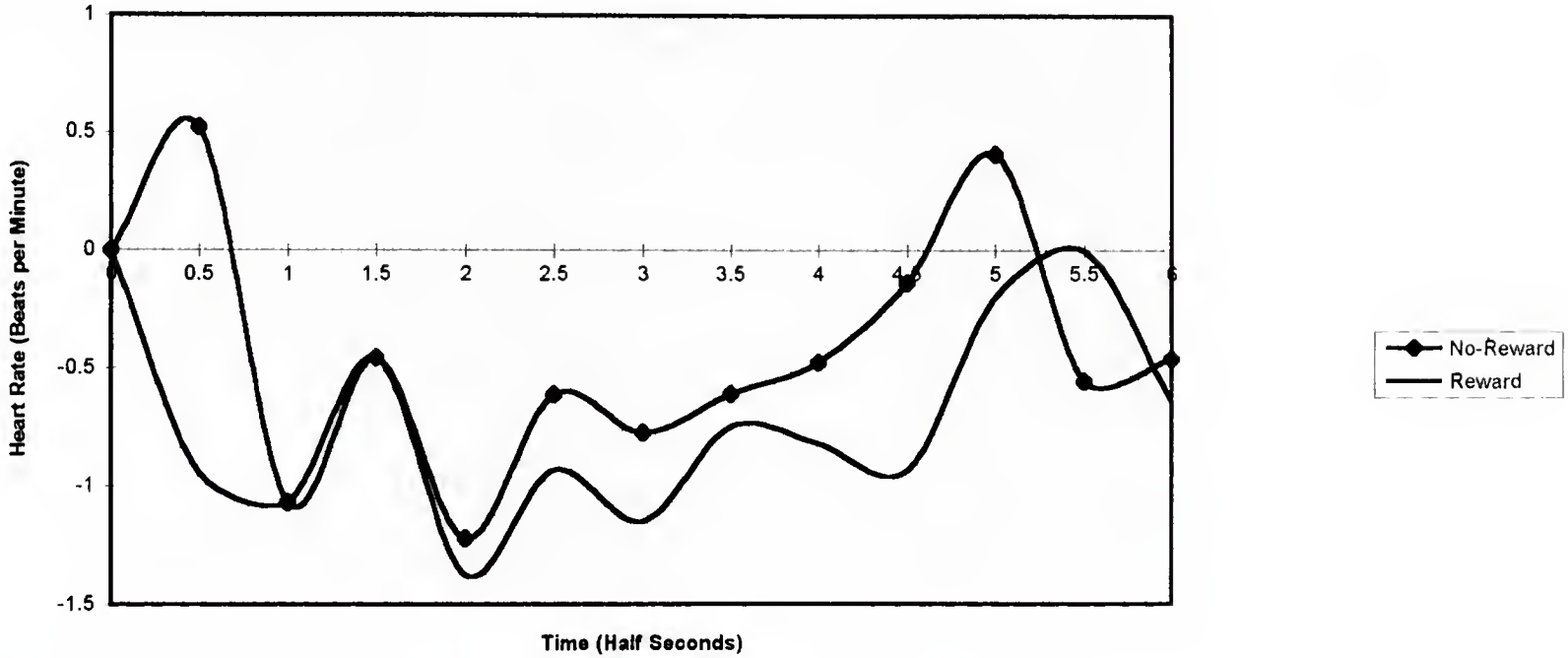

Figure C-6 Heart Rate Change Scores in LHD Ss during Reward Task

Table C-40 ANOVA Table of Mean HR Change from Baseline during the Reward Task

	SS	DF	MS	F	SIG of F
Group	2.94891	3	.98297	.76216	.5215
Subject(Group)	55.45809	43	1.28972		
Tone	1.55279	1	1.55279	.96105	.3324
Tone by Group	2.06203	3	.68734	.42541	.7358
Tone by Subject (Group)	69.47577	43	1.61572		

Table C-41 ANOVA Table of D1 during the Reward Task

	SS	DF	MS	F	SIF of F
Group	75.56907	3	25.18969	1.3618	.2672
Subject(Group)	795.37279	43	18.49704		
Tone	.00002	1	.00002	.00000	.9988
Tone by Group	10.11084	3	3.37028	.38576	.7638
Tone by Subject(Group)	375.67775	43	8.73669		
Block	8.88889	3	2.96296	.45990	.7108
Block by Group	37.20585	9	4.13398	.64166	.7596
Block by Subject(Group)	831.09682	129	6.44261		
Tone by Block	8.23695	3	2.74565	.54872	.6499
Tone by Block by Group	32.81069	9	3.64563	.72859	.6820
Tone by Block by Subject(Group)	645.47852	129	5.00371		

Table C-42 ANOVA Table of A1 during the Reward Task

	SS	DF	MS	F	P
Group	46.60895	9	15.53632	.66432	.5786
Subject(Group)	1005.63880	43	23.38695		
Tone	10.35746	1	10.35746	.79977	.3761
Tone by Group	23.66528	3	7.88843	.60912	.6127
Tone by Subject(Group)	556.87177	43	12.95051		
Block	16.50844	3	5.50281	.63107	.5962
Block by Group	25.22869	3	2.80319	.32148	.9667
Block by Subject(Group)	1124.84796	129	8.71975		
Tone by Block	4.64534	3	1.54845	.19863	.8972
Tone by Block by Group	47.42106	9	5.26901	.67590	.7295
Tone by Block by Subject(Group)	1005.62893	129	7.79557		

Table C-43 ANOVA Table of D2 during the Reward Task

	SS	DF	MS	F	SIF of F
Group	47.90806	3	15.9694	.85091	.4738
Subject(Group)	80699422	43	18.7673		
Tone	4.18219	1	4.18219	.40701	.5269
Tone by Group	9.15995	3	3.05332	.29715	.8272
Tone by Subject(Group)	441.84627	43	10.2755		
Block	34.85478	3	11.6183	1.8012	.1502
Block by Group	53.03792	9	5.89310	.91366	.5156
Block by Subject(Group)	832.04873	129	6.44999		
Tone by Block	15.82781	9	5.27594	.72497	.5389
Tone by Block by Group	48.26175	9	5.36242	.73685	.6745
Tone by Block by Subject(Group)	938.79032	129	7.27744		

Table C-44 ANOVA Table of Percentage of SCR Responses during the Reward Task

	SS	DF	MS	F	SIG of F
Group	1267.493	3	422.498	.4776	.6996
Subject(Group)	38042.614	43	884.712		
Trial	26.397	1	26.397	.5624	.4574
Trial by Group	261.948	3	87.316	1.8602	.1507
Trial by Subject(Group)	2018.371	43	46.939		

Table C-45 ANOVA Table of Recoded Range Corrected SCR during the Reward Task

	SS	DF	MS	F	SIG of F
Group	1587.75565	3	529.25188	.6911	.5625
Subject(Group)	32929.5129	43	765.80263		
Block	872.80795	3	290.93598	2.841	.0405
Block by Group	367.05610	3	40.78401	.3983	.9340
Block by Subject(Group)	13210.1142	129	102.40399		
Tone	38.93401	1	38.93401	.4692	.4970
Tone by Group	49.28116	3	16.42705	.1980	.8972
Tone by Subject(Group)	3568.1784	43	82.98089		
Block by Tone	839.36650	3	279.7888	3.316	.0221
Block by Tone by Group	590.95788	9	65.66199	.7781	.6369
Block by Tone by Subject(Group)	10886.243	129	84.38948		

Table C-46 T-Tests of Block Differences in Recoded Range Corrected SCR during the Reward Task

Block	Mean Diff.	DF	T-Value	P-Value
Block 1, Block 2	3.823	46	2.727	.0090
Block 1, Block 3	3.492	46	1.923	.0607
Block 1, Block 4	2.800	46	1.432	.1588
Block 2, Block 3	-.332	46	-.328	.7446
Block 2, Block 4	-1.023	46	-.899	.3732
Block 3, Block 4	-.691	46	-.643	.5236

Table C-47 T-Tests of Condition Differences in Recoded Range Corrected SCR by Block during the Reward Task

Blocks	Mean Diff.	DF	T-Value	P-Value
Block 1	5.681	46	3.172	.0027
Block 2	-1.039	46	-.654	.5163
Block 3	-2.024	46	-1.199	.2365
Block 4	-.140	46	-.060	.9522

Table C-48 ANOVA Table of Corrugator EMG during the Reward Task

	SS	DF	MS	F	SIG of F
Group	.233	3	.078	.800	.5003
Subject(Group)	4.265	44	.097		
Block	.098	3	.033	1.067	.3654
Block by Group	.388	3	.043	1.401	.1940
Block by Subject(Group)	4.060	132	.031		
Tone	.020	1	.020	.397	.5318
Tone by Group	.098	3	.028	.558	.6454
Tone by Subject(Group)	2.210	44	.050		
Block by Tone	.098	3	.029	.558	.6170
Block by Tone by Group	.602	9	.067	1.397	.1956
Block by Tone by Subject(Group)	6.319	132	.048		

Table C-49 ANOVA Table of Left-Sided Zygomatic EMG during Reward Task

	SS	DF	MS	F	SIG of F
Group	.300	3	.100	1.783	.1644
Subject(Group)	2.473	44	.056		
Block	.063	3	.021	.688	.5612
Block by Group	.395	9	.044	1.432	.1807
Block by Subject(Group)	4.047	132	.031		
Tone	.018	1	.018	.687	.4118
Tone by Group	.021	3	.007	.270	.8466
Tone by Subject(Group)	1.128	44	.026		
Block by Tone	.075	3	.025	.757	.5202
Block by Tone by Group	.216	9	.026	.731	.6799
Block by Tone by Subject(Group)	4.332	132	.033		

Table C-50 ANOVA Table of Right-sided Zygomatic EMG during the Reward Task

	SS	DF	MS	F	SIG of F
Group	.063	3	.021	.410	.7469
Subject(Group)	2.251	44	.051		
Block	.041	3	.014	.623	.5975
Block by Group	.271	9	.030	1.388	.1996
Block by Subject(Group)	2.867	132	.022		
Tone	.001	1	.001	.080	.7782
Tone by Group	.032	3	.011	1.404	.2543
Tone by Subject(Group)	.337	44	.008		
Block by Tone	.001	3	.001	.023	.9952
Block by Tone by Group	.107	9	.012	1.063	.3947
Block by Tone by Subject(Group)	1.473	132	.011		

Table C-51 Kruskal-Wallis Tests of SAM Ratings during Reward Task

			Corrected for Ties	
	Chi-Square	Significance	Chi-Square	Significance
Valence (Reward)	1.4598	.6916	2.2787	.5166
Valence (Control)	.4543	.9288	.4623	.9271
Arousal (Reward)	5.9702	.1131	6.3427	.0961
Arousal (Control)	1.1635	.7618	1.2536	.7002
Dominance (Reward)	1.0096	.7989	1.0466	.7082
Dominance (Control)	.8140	.8461	1.0466	.7900

Table C-52 ANOVA Table of Mean HR Change from Baseline comparing Shock and Reward Tasks

	SS	DF	MS	F	SIG of F
Group	5.663	3	1.888	1.060	.3758
Subject(Group)	76.542	43	1.780		
Condition	.919	1	.919	.237	.6289
Condition by Group	4.656	43	1.552	.400	.7537
Condition by Subject(Group)	166.798	43	3.879		

Table C-53 ANOVA Table of D1 comparing Shock and Reward Tasks

	SS	DF	MS	F	SIG of F
Group	21.623	3	7.208	.7057	.5539
Subject(Group)	439.211	43	10.214		
Block	11.793	3	3.930	.3313	.8028
Block by Group	140.416	9	15.608	1.3148	.2353
Block by Subject(Group)	1530.77	129	11.866		
Condition	.153	1	.153	.0100	.9208
Condition by Group	32.091	3	10.697	.6971	.5589
Condition by Subject(Group)	659.807	43	15.344		
Block by Condition	24.706	3	8.265	.9892	.4002
Block by Condition by Group	43.727	9	4.859	.5836	.8087
Block by Condition by Subject(Group)	1074.00	129	8.326		

Table C-54 ANOVA Table of A1 comparing Shock and Reward Tasks

	SS	DF	MS	F	SIG of F
Group	98.429	3	32.810	1.667	.1882
Subject(Group)	846.152	43	19.678		
Block	3.623	3	1.208	.0730	.9744
Block by Group	203.068	9	22.563	1.363	.2116
Block by Subject(Group)	2135.53	129	16.555		
Condition	41.456	1	41.456	1.395	.2440
Condition by Group	172.971	3	57.657	1.941	.1373
Condition by Subject(Group)	1277.51	43	29.710		
Block by Condition	30.398	3	10.133	.773	.5114
Block by Condition by Group	200.119	9	22.235	1.695	.0964
Block by Condition by Subject(Group)	1691.90	129	13.115		

Table C-55 ANOVA Table of D2 comparing Shock and Reward Tasks

	SS	DF	MS	F	SIG of F
Group	58.169	3	19.390	1.373	.2639
Subject(Group)	607.428	43	14.126		
Block	21.842	3	7.281	.5564	.6448
Block by Group	153.363	9	17.040	1.3021	.2419
Block by Subject(Group)	1688.14	129	13.087		
Condition	.1089	1	.109	.0037	.9517
Condition by Group	68.835	3	22.944	.7807	.5113
Condition by Subject(Group)	1263.85	43	29.392		
Block by Condition	20.387	3	6.796	.6272	.5987
Block by Condition by Group	50.065	9	5.563	.5134	.8627
Block by Condition by Subject(Group)	1397.76	129	10.835		

Table C-56 ANOVA Table of Percentage of SCR Responses comparing Shock and Reward Tasks

	SS	DF	MS	F	Sig of F
Group	2764.21	3	921.40	6.53	.0010
Subject(Group)	6063.45	43	141.01		
Condition	1996.37	1	1996.37	16.50	.0002
Condition by Group	1021.63	3	340.54	2.81	.0504
Condition by Subject(Group)	5202.84	43	121.00		

Table C-57 T-Tests of Group Differences in Percentage of Responses for Recoded Range Corrected SCR during the Shock and Reward Tasks Combined

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-9.583	34	-2.861	.0072
LHD, RHD	1.477	21	.776	.4465
RHD, CONS	11.061	33	3.282	.0024

Table C-58 T-Tests of Percentage of Responses for Recoded Range Corrected SCR during the Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-17.50	34	-.3443	.0015
LHD, RHD	-2.803	21	-1.000	.3285
RHD, CONS	14.697	33	2.814	.0082

Table C-59 T-Tests of Percentage of Responses for Recoded Range Corrected SCR during the Reward Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-1.667	34	-.461	.6475
LHD, RHD	5.758	21	2.158	.0426
RHD, CONS	7.424	33	1.882	.0687

Table C-60 ANOVA Table of Recoded Range Corrected SCR comparing Shock and Reward Tasks

	SS	DF	MS	F	SIG of F
Group	2351.40	3	783.805	3.07	.0377
Subject(Group)	10976.56	43	255.27		
Block	595.58	3	198.52	1.33	.2670
Block by Group	1018.28	3	113.14	.7588	.6545
Block by Subject(Group)	19234.29	129	149.10		
Condition	3573.57	1	3573.57	23.21	.0001
Condition by Group	1335.61	3	445.20	2.89	.0462
Condition by Subject(Group)	6619.34	43	153.94		
Block by Condition	4598.28	3	1532.76	9.18	.0001
Block by Condition by Group	1831.17	9	203.46	1.22	.2891
Block by Condition by Subject(Group)	21536.40	129	166.95		

Table C-61 T-Tests of Group Differences in Recoded Range Corrected SCR during Shock and Reward Tasks Combined

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-5.732	34	-2.285	.0287
LHD, RHD	1.310	21	1.029	.3154
RHD, CONS	7.042	33	2.733	.0100

Table C-62 T-Tests of Block Differences in Recoded Range Corrected SCR comparing the Shock and Reward Tasks

	Mean Diff.	DF	T-Value	P-Value
Shock Block1, Reward Block1	17.027	46	4.724	<.0001
Shock Block 2, Reward Block 2	3.712	46	1.586	.1195
Shock Block 3, Reward Block 3	1.065	46	.433	.6671
Shock Block 4, Reward Block 4	7.961	46	2.925	.0053

Table C-63 T-Tests of Recoded Range Corrected SCR during Shock Task

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-10.699	34	-2.809	.0082
LHD, RHD	1.608	21	.721	.4790
RHD, CONS	12.306	33	3.234	.0028

Table C-64 T-Tests of Recoded Range Corrected SCR during Reward Tasks

	Mean Diff.	DF	T-value	P-value
LHD, CONS	-.765	34	-.321	.7503
LHD, RHD	1.012	21	.592	.5604
RHD, CONS	1.777	33	.686	.4973

Table C-65 ANOVA Table of Corrugator EMG compairng Shock and Reward Tasks

	SS	DF	MS	F	Sig of F
Group	.1982	3	.066	.7044	.5545
Subject(Group)	4.128	44	.094		
Condition	.009	1	.009	.3418	.5618
Condition by Group	.051	3	.017	.6437	.5911
Condition by Subject(Group)	1.159	44	.026		
Block	.035	3	.012	.1982	.8975
Block by Group	.786	9	.087	1.486	.1535
Block by Subject(Group)	7.761	132	.059		
Condition by Block	.146	9	.049	.8974	.4445
Condition by Block by Group	.433	9	.048	.8854	.5402
Condition by Block by Subject(Group)	7.164	132	.054		

Table C-66 ANOVA Table of Left-sided Zygomatic EMG compairng Shock and Reward Tasks

	SS	DF	MS	F	Sig of F
Group	.054	3	.019	.5827	.6295
Subject(Group)	1.372	44	.031		
Condition	.001	1	.001	.0271	.8700
Condition by Group	.051	3	.017	.4874	.6928
Condition by Subject(Group)	1.530	44	.035		
Block	.118	3	.039	.7502	.5241
Block by Group	.167	3	.019	.3546	.9542
Block by Subject(Group)	6.906	132	.052		
Condition by Block	.405	3	.015	.3799	.7676
Condition by Block by Group	.405	9	.045	1.152	.3314
Condition by Block by Subject(Group)	5.157	132	.039		

Table C-67 ANOVA Table of Right-sided Zygomatic EMG compairng Shock and Reward Tasks

	SS	DF	MS	F	Sig of F
Group	.057	3	.019	1.428	.2473
Subject(Group)	.585	44	.013		
Condition	.003	1	.003	.204	.6534
Condition by Group	.109	3	.039	2.211	.1002
Condition by Subject(Group)	.722	44	.016		
Block	.027	3	.009	.385	.7640
Block by Group	.233	9	.026	1.101	.3667
Block by Subject(Group)	3.107	132	.024		
Condition by Block	.039	3	.013	.625	.6004
Condition by Block by Group	.183	9	.020	.990	.4517
Condition by Block by Subject(Group)	2.716	132	.021		

Table C-68 Kruskal-Wallis Tests of SAM Ratings comparing Shock and Reward Tasks

			Corrected for Ties	
	Chi-Square	Significance	Chi-Square	Significance
Valence (Shock-Control)	1.7674	.6220	1.8007	.6148
Valence (Reward-Control)	.6909	.8753	.7043	.8722
Arousal (Shock-Control)	1.2634	.7378	1.3071	.7274
Arousal (Reward-Control)	4.4192	.2196	5.1711	.1597
Dominance (Shock-Control)	3.3178	.3452	3.8878	.2738
Dominance (Reward-Control)	.5085	.9170	.8345	.8412

Table C-69 ANCOVA Table of Percentage of SCR Responses during the Shock Task with Medication as a Covariate

	SS	DF	SS	F	Sig of F
Within and Residual (Group)	55258.13	42	1315.67		
Regression	544.14	1	544.14	.41	.524
Group	8057.17	3	2685.72	2.04	.123
Within and Residual (Tone)	3614.77	43	84.06		
Tone	2481.89	1	2481.89	29.52	.000
Tone by Group	1630.97	3	543.66	6.47	.001

Table C-70 ANCOVA Table of Recoded Range Corrected SCR during the Shock Task with Medication as a Covariate

	SS	DF	MS	F	Sig of F
Within and Residual (Group)	34779.18	42	828.08		
Regression	723.56	1	723.56	.87	.355
Group	3888.28	3	1296.09	1.57	.212
Within and Residual (Block by Group)	12196.85	129	94.55		
Block	3987.68	3	1329.23	14.06	.000
Group by Block	1123.81	9	124.87	1.32	.232
Within and Residual (Tone by Group)	7716.13	43	179.44		
Tone	4191.21	1	4191.21	23.36	.001
Group by Tone	3551.95	3	1183.98	6.60	.001
Within and Residual (Block by Tone by Group)	10495.21	129	81.36		
Block by Tone	934.60	3	311.53	3.83	.011
Group by Block by Tone	355.22	9	39.47	.49	.882

Table C-71 ANCOVA Table of Percentage of SCR Responses comparing Shock and Reward Tasks with Medication as a Covariate

	SS.	DF	SS	F	Sig of F
Within and Residual (Group)	6045.49	42	143.94		
Regression	17.95	1	17.95	.12	.726
Group	2266.16	3	755.39	5.25	.004
Within and Residual (Tone)	5202.84	43	121.00		
Tone	1996.37	1	1996.37	16.50	.000
Tone by Group	1021.63	3	340.54	2.81	.050

Table C-72 ANCOVA Table of Recoded Range Corrected SCR comparing Shock and Reward Tasks with Medication as a Covariate

	SS	DF	MS	F	Sig of F
Within and Residual (Group)	13749.50	42	327.37		
Regression	92.04	1	92.04	.28	.599
Group	3748.05	3	1249.35	3.82	.017
Within and Residual (Block by Group)	21210.09	129	164.42		
Block	132.40	3	44.13	.27	.848
Group by Block	586.50	9	65.17	.40	.935
Within and Residual (Tone by Group)	8727.08	43	202.96		
Tone	5038.05	1	5038.05	24.82	.000
Group by Tone	2929.75	3	976.58	4.81	.006
Within and Residual (Block by Tone by Group)	21552.83	129	167.08		
Block by Tone	3415.53	3	1138.51	6.81	.000
Group by Block by Tone	1305.85	9	145.09	.87	.555

Table C-73 ANOVA Table of Positive Affect during Shock Task of Experiment Two

	SS	DF	MS	F	SIG of F
Group	458.69792	3	152.89931	.74276	.5323
Subject(Group)	9057.54167	44	205.85322		
Trial	1.76042	1	1.76042	.19338	.6623
Trial by Group	15.19792	3	5.06597	.55650	.6465
Trial by Subject(Group)	400.54167	44	9.10322		

Table C-74 ANOVA Table of Negative Affect during Shock Task of Experiment Two

	SS	DF	MS	F	SIG of F
Group	39.58333	3	13.19444	.39313	.7585
Subject(Group)	1476.75000	44	33.56250		
Trial	24.00000	1	24.00000	9.5207	.0035
Trial by Group	6.08333	3	2.02778	.80441	.4982
Trial by Subject(Group)	110.91667	44	2.52083		

Table C-75 Kruskal-Wallis Tests of SAM Ratings during Shock Task of Experiment Two

			Corrected for Ties	
	Chi-Square	Significance	Chi-Square	Significance
Valence (Shock)	1.0028	.8006	1.0581	.7872
Valence (Control)	2.5091	.4736	3.2613	.3531
Arousal (Shock)	1.4768	.6876	1.6324	.6521
Arousal (Control)	5.2128	.1569	8.6035	.0351
Dominance (Shock)	4.1869	.2420	5.9760	.1128
Dominance (Control)	1.2881	.7320	3.0704	.3809

Table C-76 Mann-Whitney U, Wilcoxon Rank Sum W Tests of Arousal Ratings during the No-Shock Condition of the Shock Task of Experiment Two

				Corrected for Ties	
	U	W	P-Value	Z	Significance
LHD, LH NCS	42.5	179.5	.0887	-2.1239	.0337
LHD, RHD	71.5	118.5	.9774	-.0602	.9520
LHD, RH NCS	50.5	171.5	.2189	-1.6441	.1001
LH NCS, RHD	40.5	118.5	.0684	-2.2718	.0231
LH NCS, RH NCS	58.5	136.5	.4428	-.8686	.3851
RHD, RH NCS	48.0	174.0	.1782	-1.8459	.0649

Table C-77 ANOVA Table of Positive Affect during the Reward Task of Experiment Two

	SS	DF	MS	F	SIG of F
Group	225.19068	3	75.06356	.49119	.6902
Subject(Group)	6571.23485	43	152.81942		
Trial	230.83636	1	230.83636	7.5209	.0089
Trial by Group	141.83672	3	47.27891	1.5404	.2178
Trial by Subject(Group)	1319.78030	43	30.69257		

Table C-78 ANOVA Table of Negative Affect during the Reward Task of Experiment Two

	SS	DF	MS	F	SIG of F
Group	46.02345	3	15.34115	.46770	.7063
Subject(Group)	1410.46591	43	32.80153		
Trial	.12803	1	.12803	.19847	.6582
Trial by Group	5.09115	3	1.69705	2.6307	.0621
Trial by Subject(Group)	27.73864	43	.64508		

Table C-79 Kruskal-Wallis Tests of SAM Ratings during Reward Task of Experiment Two

			Corrected	for Ties
	Chi-Square	Significance	Chi-Square	Significance
Valence (Reward)	1.2277	.7464	2.4519	.4[illegible]5
Valence (Control)	.0086	.9998	.0122	.9996
Arousal (Reward)	5.3693	.1467	6.6205	.0850
Arousal (Control)	2.7838	.4262	5.0710	.1667
Dominance (Reward)	1.4177	.7014	2.8305	.4185
Dominance (Control)	.6541	.8839	2.2841	.5156

Table C-80 ANOVA Table of Positive Affect comparing Shock and Reward Tasks during Experiment Two

	SS	DF	MS	F	SIG of F
Group	150.64	3	50.21	.9899	.4066
Subject(Group)	2181.11	43	50.72		
Condition	186.88	1	186.88	6.400	.0152
Condition by Group	165.79	3	55.26	1.892	.1451
Condition by Subject(Group)	1255.53	43	29.198		

Table C-81 ANOVA Table of Negative Affect comparing Shock and Reward Tasks of Experiment Two

	SS	DF	MS	F	SIG of F
Group	12.58	3	4.19	1.24	.3063
Subject(Group)	145.25	43	3.38		
Condition	26.49	1	26.49	8.64	.0053
Condition by Group	10.04	3	3.35	1.09	.3629
Condition by Subject(Group)	131.79	43	3.06		

Table C-82 Kruskal-Wallis Tests of SAM Ratings comparing Shock and Reward Tasks of Experiment Two

			Corrected for Ties	
	Chi-Square	Significance	Chi-Square	Significance
Valence (Shock-Control)	.5716	.9029	.64459	.8862
Valence (Reward-Control)	.5996	.8965	.8825	.8296
Arousal (Shock-Control)	4.3484	.2262	4.9789	.1733
Arousal (Reward-Control)	3.9173	.2705	5.2062	.1573
Dominance (Shock-Control)	2.5942	.4585	4.4929	.2129
Dominance (Reward-Control)	.6716	.8799	1.9981	.5728

REFERENCES

Ahern, G. L., & Schwartz, G. E. (1979). Differential lateralization for positive versus negative emotions. Neuropsychologia, 17, 693-698.

Ahern, G. L. & Schwartz, G. L. (1985). Differential lateralization for positive and negative emotions in the human brain: EEG spectral analysis. Neuropsychologia, 23, 745-756.

Anderson, S. W., Damasio, H., & Tranel, D. (1990). Neuropsychological impairments associated with lesions caused by tumor or stroke. Archives of Neurology, 47, 397-405.

Annett, M. (1970). A classification of hand preference by association analysis. British Journal of Psychology, 61, 303-321.

Arnold, M. (1960). Emotions and personality, (Vols 1,2). New York: Columbia University Press.

Babinski, J. (1914). Contribution a l'etude des troubles mentaux dans l'hemisplegie organique cerbrale (anosognosie). Revue Neurologique, 27, 845-848.

Bankart, C. P., & Elliot, R. (1974). Heart rate and skin conductance in anticipation of shocks with varying probability of occurence. Psychophysiology, 11, 160-174.

Blonder, L. X., Bowers, D., Heilman, K. M. (1991). The role of the right hemisphere on emotional communication. Brain, 114, 1115-1127.

Blonder, L. X., Burns, A., Bowers, D., Moore, R., Heilman, K. M. (1992). Right hemisphere expressivity during natural conversation. Journal of Clinical & Experimental Neuropsychology, 13, 85.

Borod, J. Emotional Expression and Brain Mechanisms: Lateralization of Behavior in Brain-Damaged and Normal Adults. Submitted for publication).

Borod, J. C., Andelman, F., Obler, L. K., Tweedy, J. R., & Welkowitz, J. (1992). Right hemisphere specialization for the identification of emotional words and sentences: Evidence from stroke patients. Neuropsychologia.

Borod, J. & Koff, E. (1990). Lateralization for facial emotion behavior: A methodological perspective. International Journal of Psychology, 25, 157-177.

Borod, J., Koff, E., Lorch, M., & Nicholas, M. (1985). Channels of emotional expression in patients with unilateral brain damage. Archives of Neurology, 42, 345-348.

Borod, J., Koff, E., Perlman-Lorch, J., Nicolas, M. (1986). The expression and perception of facial emotions in brain damaged patients. Neuropsychologia, 24, 169-180.

Borod, J., Koff, E., Perlman-Lorch, M., Nicolas, M., & Welkowitz, J. (1988). Emotional and nonemotional facial behavior in patients with unilateral brain damage. Journal of Neurology, Neurosurgery, and Psychiatry, 51, 826-832.

Borod, J. C., Koff, E., & White, B. (1983). Facial asymmetry in spontaneous and posed expressions of emotion. Brain and Cognition, 2, 165-175.

Bowers, D., Bauer, R. M., Coslett, H. B., & Heilman, K. M. (1985). Processing of faces by patients with unilateral hemispheric lesions. Dissociation between judgements of facial affect and facial identity. Brain and Cognition, 4, 258-272.

Bowers, D, Blonder, L. X., Feinberg, T., & Heilman, K. M. (1991). Differential impact of right and left hemisphere lesions on facial emotion and object imagery. Brain, 114, 1-17.

Bowers, D., Coslett, H. B., Bauer, R. M., Speedie, L. J., Heilman, K. M. (1987). Comprehension of emotional prosody following unilateral hemispheric lesions: Processing defect vs. distraction defect. Neuropsychologia, 25, 317-328.

Bowers, K. S. (1971a). The effects of UCS temporal uncertainty on heart rate and pain. Psychophysiology, 8, 382-389.

Bowers, K. S. (1971b). Heart rate and GSR concomitants of vigilance and arousal. Canadian Journal of Psychology, 25, 175-184.

Bradley, M. M., Lang, P. J., Cuthbert, B. N. (1991). The Gainesville murders: imagining the worst. Society for Psychological Research, 28, s14 [abstract].

Briggs, G. G., & Nebes, R. D. (1975). Patterns of hand preference in a student population. Cortex, 11, 230-238.

Brown, S. L., & Schwartz, G. E. (1980). Relationships between facial electromyography and subjective experience during affective imagery. Biological Psychology, 11, 49-62.

Brownell, H., Michelon, D., Powelson, J., Gardner, H. (1983). Surprise but not coherence: Sensitivity to verbal humor in right hemisphere patients. Brain and Language, 18, 20-27.

Buck, R. (1990). Using FACS versus communication scores to measure spontaneous facial expression of emotion in brain damaged patients. Cortex, 26, 275-280.

Buck, R., & Duffy, R. J. (1980). Nonverbal communication of affect in brain damaged patients. Cortex, 16, 351-362.

Cacioppo, J. T., Martzke, J. S., Petty, R. E., & Tassinary, L. G. (1988). Specific forms of facial EMG response index emotions during an interview: from darwin to the continuous flow hypothesis of affect-laden information processing. Journal of Personality and Social Psychology, 54, 592-604.

Cacioppo, J. T., & Petty, R. E., Losch, M. E., & Kim, H. S. (1986). Electromyography activity over facial muscle regions can differentiate the valence and intensity of affective reactions. Journal of Personality and Social Psychology, 50, 260-268.

Cacioppo, J. T., Petty, R. E., Morris, K. J. (1985). Semantic,evaluative, and self-referent processing: Memory, cognitive effort, and somatovisceral activity. Psychophysiology, 22, 371-384.

Cacioppo, J. T. & Tassinary, L. G (1990). Psychophysiology and psychophysiological inference. In J. T. Cacioppo & L. G. Tassinary (Eds.), Principles of psychophysiology. New York: Cambridge University Press.

Caltagirone, C., Ekman, P., Friesen, W. V., Gainotti, G., Mammucari, A., Pizzamiglio, L., & Zoccolatti, P. (1989). Posed emotional facial expressions in brain damaged patients. Cortex, 25, 653-663.

Campbell, R. (1978). Asymmetries in interpreting and expressing a posed facial expression. Cortex, 14, 327-342.

Cancelliere, A. B. & Kertesz, A. (1990). Lesion localization in acquired deficits of emotional expression and comprehension. Brain and Cognition, 13, 133-147.

Cannon, W. B. (1927). The James-Lange theory of emotion: a critical examination and alternative theory. American Journ of Psychology, 39, 106-124. Reprinted in M. Arnold (Ed.), The Nature of Emotion. Baltimore: Penguin, 1968.

Cicone, M., Wapner, W., & Gardner, H. (1980). Sensitivity to emotional expressions and situations in organic patients. Cortex, 16, 145-158.

Damasio, H. Y Damasio, A. R. (1989). Lesion analysis in neuropsychology. New York: Oxford University Press.

Darwin, C. (1872/1965). The expression of emotion in man and animals, Chicago: The University of Chicago Press. (Original work published 1872).

Davidson, R. J. (1985) Affect, cognition, and hemisphericspecialization. In C. E. Izard, J. Kagan, & R. B. Zajonc (Eds.), emotions, cognition, and behavior. Cambridge: Cambridge University Press.

Davidson, R. J. Parsing affective space: Perspectives from neuropsychology and psychophysiology. Submitted for publication.

Davidson, R., Ekman, P., Saron, C., Senulis, J., & Friesen, W. (1990). Approach/withdrawal and cerebral asymmetry. Emotional expression and brain physiology. Journal of Personality and Social Psychology, 56, 330-341.

Davidson, R. J. & Fox , N. A. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in ten month old infants. Science, 218, 1235-1237.

Davidson, R. J. & Schwartz, G. E. (1976). Patterns of cerebral lateralization during cardiac biofeedback

versus the self-regulation of emotion: Sex differences. Psychophysiology, 16, 202-203.

Davidson, R. J., Schwartz, G. E., Saron, C., Bennett, J., & Goldman, D. J. (1979). Frontal versus parietal EEG asymmetry during positive and negative affect. Psychophysiology, 16, 202-203.

Deane, G. E. (1961). Human heart rate responses during experimentally induced anxiety. Journal of Experimental Psychology, 61, 489-493.

Deane, G. E. (1969). Cardiac activity during experimentally induced anxiety. Psychophysiology, 6, 17-30.

DeKosky, S., Heilman, K. M., Bowers, D., & Valenstein, E. (1980). Recognition and discrimination of emotional faces and pictures. Brain and Language, 9, 206-214.

Denny-Brown, D., Meyer, J. S., & Horenstein, S. (1952). The significance of perceptual rivalry resulting from parietal lesions. Brain, 75, 434-471.

Dimberg, U. & Lundquist, L. (1990). Gender differences in facial reactions to facial expressions. Biological Psychology, 30, 151-159.

Duda, P. & Brown, J. (1984). Lateral asymmetry of positive and negative emotions. Cortex, 20, 253-261.

Duffy, E. (1957). The psychological significance of the concept of "arousal" and "activation." Psychological Review, 64, 265-275.

Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. Journal of Personality and Social Psychology, 17, 124-129.

Ekman, P., & Friesen, W. V. (1978). The facial action coding system (FACS): A technique for the measurement of facial action. Palo Alto, CA: Consulting Psychologists Press.

Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. Science, 221, 1208-1210.

Elliot, R. (1966). Effects of uncertainty about the nature and advent of a noxious stimulus (shock) upon heart rate. Journal of Personality and Social Psychology, 3, 353-356.

Ellsworth, P. C. & Smith, C. E. (1988). Shades of joy: Appraisals differntiating among positive emotions. Emotions and Cognition, 2, 301-331.

Erlichman, H., & Weinberger, A. (1978). Lateral eye movements and hemispheric asymmetry: A critical review. Psychological Bulletin, 85, 1080-1101.

Etcoff, N. (1984). Perceptual and conceptual organization of facial emotions. Brain and Cognition, 3, 385-412.

Ferguson, G. A. (1971). Statistical Analysis in Psychology & Education. New York: McGraw-Hill.

Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational apprach. Psychophysiology, 25, 373-391.

Fridlund, A. J., & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. Psychophysiology, 23, 567-589.

Fridlund, A., Ekman, P., & Oster, H. (1987). Facial expressions of emotion: Review of literature, 1970-1983. In A. Siegman & S. Feldstein (Eds.), Nonverbal behavior and communication. Hillsdale, NJ: Lawrence Erlbaum.

Fox, N. A., & Davidson, R. J. (1984). Hemispheric substrates of affect: A developmental model. In: N. A. Fox & R. J. Davidson (Eds.), The psychobiology of affective development. Hillsdale, NJ: Erlbaum.

Gainotti, G. (1972). Emotional behavior and hemispheric side of the lesion. Cortex, 8, 41-55.

Gardner, H., Brownell, H. H., Wapner, W., & Michelon, D. (1983). Missing the point: The role of the right hemisphere in the processing of complex linguistic materials. In E. Perceman (Ed.), Cognitive processing in the right hemisphere. New York: Academic.

Gardner, H., Ling, P. K., Flam, I., & Silverman, J. (1975). Comprehension and appreciation of humorous material following brain damage. Brain, 98, 399-412.

Goldstein, K. (1948). Language and language disturbances. New York: Grune & Stratton.

Gorelick, P. B., & Ross, E. D. (1987). The aprosodias: Further functional-anatomical evidence for the organization of affective language in the right

hemisphere. Journal of Neurology, Neurosurgery, and Psychiatry, 50, 553-560.

Graham, F. K., & Clifton, R. K. (1966).Heart rate changes as a component of the orienting response. Psychological Bulletin, 65, 305-320.

Graves, R., Landis, T., & Goodlass, H. (1981). Laterality and sex differences for visual recognition of emotional and nonemotional words. Neuropsychologia, 19, 95-102.

Greenwald, M. K., Cook, E. W., & Lang, P. J. (1989). Affective judgment and psychophysiological response: dimensional covariation in the evaluation of picture stimuli. Journal of Psychophysiology, 3, 51-64.

Haggard, M. P., & Parkinson, A. M. (1971). Stimulus and task factors as determinants of ear advantages. Quarterly Journal of Experimental Psychology, 23, 168-177.

Hare. R. D. (1972). Cardiovascular components of orienting and defensive responses. Psychophysiology, 9, 606-614.

Hare, R. D. (1973). Orienting and defensive responses to visual stimuli. Psychophysiology, 10, 453-464.

Heilman, K. M., Bowers, D., Speedie, L., & Coslett, B. (1984). Comprehension of affective and nonaffective speech. Neurology, 34, 917-921.

Heilman, K. M., Bowers, D., & Valenstein, E. (1985). Emotional disorders associated with neurologic disease. In K. Heilman & E. Valenstein (Eds.). Clinical Neuropsychology (2nd Ed). New York: Oxford Press

Heilman, K. M., Bowers, D., & Valenstein, E. (1993). Emotional disorders associated with neurologic disease. In K. Heilman & E. Valenstein (Eds.). Clinical Neuropsychology (3rd Ed). New York: Oxford Press

Heilman, K. M., Scholes, R., & Watson, R. T. (1975). Auditory affective agnosia: Disturbed comprehension of affective speech. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 69-72.

Heilman, K. M., Schwartz, H. D., & Watson, R. T. (1978). Hypoarousal in patients with the neglect syndrome and emotional indifference. Neurology, 28, 229-232.

Heilman, K. M., Watson, R. T., & Bowers, D. (1983). Affective disorders associated with hemispheric disease. In:K. M. Heilman (Ed.), Neuropsychology of human emotion. New York: Guilford Press.

Heilman, K. M., Watson, R. T., & Valenstein, E. (1993). Neglect and related disorders. In K. Heilman & E. Valenstein (Eds.). Clinical Neuropsychology (3rd Ed). New York: Oxford Press

Heller, W. (1990). The Neuropsychology of emotion: Developmental patterns and implications for psychopathology. In Psychological and Biological Approaches to Emotion. N. L. Stein, B. Leventhal, & T. Trebasso (Eds.). Lawrence Erlbaum: Hillsdale, N.Y.

Heller, W. & Levy. J. (1981). Perception and expression of emotion in right handers and left handers. Neuropsychologia, 19, 263-272.

Hodes, R. L., Cook, E. W., & Lang, P. J. (1985). Individual differences in autonomic response: Conditioned association or conditioned fear? Psychophysiology, 22, 545-560.

Hodges, W. F. & Spielberger, C. D. (1966). The effects of threat of shock on heart rate for subjects who differ in manifest anxiety and fear of shock. Psychophysiology, 2, 287-294.

House, A., Dennis, M., Warlow, C., Hawton, K., & Molyneux, A. (1990). Mood disorders after stroke and their relation to lesion location. Brain, 113, 1113-1129.

Izard, C. E. (1977). Human emotions. New York: Plenum Press.

Izard, C.E. (1978). The Maximally Discriminative Facial Movement Coding System (MAX). Newark: Instructional Resources Center, University of Delaware.

James, W. (1884). What is emotion? Mind, 9, 188-205. Reprinted in K. Dunlap, Psychology Classics, Vol. 1: The emotions. Baltimore: Williams & Wilkins Company, 1922.

Jensen, R. & Fuglsang-Frederiksen, A. (1994). Qualtitative surface EMG of pericranial muscles. Relation to age and sex in a general population. Electroencephalography ans Clincial Neurophysiology, 93, 175-183.

Kent, J., Borod, J. C., Koff, E., Welkowitz, J., & Alpert, M. (1988). Posed facial emotional expression in brain-damaged patients. International Journal of Neuroscience, 43, 81-87.

Kimura, D. (1967). Functional asymmetry of the brain in dichotic listening. Cortex, 3, 163-178.

Kinsbourne, M., & Bemporad, B. (1984). Lateralization of emotion: A model and the evidence. In N. A. Fox & R. J. Davidson (Eds.), The psychobiology of affective development. Hillsdale, NJ: Erlbaum.

Kleinginna, P. R. & Kleinginna, A. M. (1981). A categorized list of emotion definitions, with suggestions for a consensual definition. Motivation and Emotion, 5, 348-379.

Klorman, R., & Ryan, R. (1980). Heart rate, contingent negative variation and evoked potentials during anticipation of affective stimulation. Psychophysiology, 17, 513-523.

Klorman, R., Wiesenfeld, A., & Austin, M. L. (1975). Autonomic responses to affective visual stimuli. Psychophysiology, 12, 553-560.

Klorman, R., Weissberg, R., & Weisenfeld, A. (1977). Individual differences in fear and autonomic reactions to affective stimulation. Psychophysiology, 4, 45-51.

Kolb, B., & Milner, B. (1981). Observations on spontaneous facial expression after focal cerebral excisions and after intracarotid injection of sodium amytal. Neuropsychologia, 19, 505-514.

Kolb, B., & Taylor, L. (1990). Neocortical substrates of emotional behavior. In N. Stein, B. Leventhal, & T. Trabasso (Eds.). Psychological and Biological Approaches to Emotion. Hillsdale, NJ: Erlbaum.

Lacey, J. I., & Lacey, B. C. (1958). The relationship of resting autonomic activity to motor impulsivity. In The Brain and Human Behavior. Baltimore: Williams & Wilkins.

Lacey, J. I. & Lacey, B. C. (1970). Some autonomic-central nervous system interrelationships. In P. Black (Ed.), Physiological correlates of emotion. New York: Academic Press.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. Psychophysiology, 16, 495-512.

Lang, P. J. (1984). Cognition in emotion: concept and action. In C. E. Izard, J. Kagan, & R. B. Zajonc (Eds.), Emotions, Cognition, and Behavior. New YorK: Cambridge University Press.

Lang, P. J. (1985). Cognition in emotion: concept and action. In C. E. Izard, J. Kagan, R. B. Zajonc (Eds.), Emotions, Cognition, and behavior. Cambridge: Cambridge University Press.

Lang, P. J., Kozak, M. J., Miller, G. A., Levin, D. N., & McLean, A., Jr. (1980). Emotional imagery: Conceptual structure & pattern of somato-visceral response. Psychophysiology, 17, 179-192.

Lang, P. J., Melamad, B. G., & Hart, J. (1970). A psychophysiological analysis of fear modification using an automated desensitization procedure. Journal of Abnormal Psychology, 76, 220-234.

Lang, P. J., Ohman, A., & Simons, R. F. (1978). The psychophysiology of anticipation. In J. Requin (Ed.), Attention and performance VII. Hillsdale, NJ: Lawrence Erlbaum.

Lange, C. G. (1922). The emotions: A psychophysiological study. In K. Dunlap (Ed.), Psychological Classics, Vol. 1: The emotions. Baltimore: Williams and Wilkins Company.

Lazarus, R. S., & Averill, J. R. (1972). Emotion and cognition: With special reference to anxiety. In C. D. Spielberger (Ed.), Anxiety: Contemporary Theory and Research. New York: Academic Press.

LeDoux, J. E. (1989). Cognitive-emotional interactions in the brain. Cognition and Emotion, 3, 267-289.

LeDoux, J. E. (1994). Emotion, memory and the brain. Scientific American, 50-57.

LeDoux, J., Sakaguchi, A., & Reis, D. J. (1984). Subcortical afferent projections of the medulla geniculate nucleus mediate emotional responses conditioned to acoustic stimuli. Journal of Neuroscience, 4, 683-698.

Leonard, C., Rolls, E., Wilson, A. (1985). Neurons in the amygdala of the monkeys with responses selective for faces. Behavioral Brain Research, 15, 159-176.

Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. Psychophysiology, 27, 363-384.

Ley, R. & Byrden, M. (1979). Hemispheric differences in processing emotions and faces. Brain and Language, 7, 127-138.

Ley, R. & Bryden, M. (1982). A dissociation of right and left hemispheric effects for recognizing emotional tone and verbal content. Brain and Cognition, 1, 3-9.

Maclean, P. D. (1949). Psychosomatic disease and the visceral brain. Recent developments bearing on the Papez theory of emotion. Psychosomatic Medicine, 11, 338-353.

Maclean, P. D. (1952). Some psychiatric implications of physiological studies of the frontotemporal portion of the limbic system. Electroencephalography and Clinical Neurophysiology., 4, 407-418.

Mammacuri, A., Caltagirone, C., Ekman, P., Friesen, W., Gainotti, G., Pizzamiglio, L, & Zoccolatti, P. (1988). Spontaneous facial expression of emotion in brain damaged patients. Cortex, 24, 521-533.

McCanne, T. R., & Anderson, J. A. (1987). Emotional responding following experimental manipulation of facial electromyographic activity. Journal of Personality and Social Psychology, 52, 759-768.

McLaren, J., & Bryson, S. (1987). Hemispheric asymmetry in the perception of emotional and neutral faces. Cortex, 23, 645-654.

Meadows, M. & Kaplan, R. F. (1992). Dissociation of autonomic and subjective responses in right hemisphere damaged patients. Presented at meeting of International Neuropsychology Society, San Diego.

Meadows, M. & Kaplan, R. F. (1994). Dissociation of autonomic and subjective responses to emotional slides in right hemisphere damaged patients. Neuropsychologia, 32, 847-856.

Miller, G. A., Levin, D. N., Kozak, M. J., Lang, P. J. (1987). Individual differences in imagery and psychophysiology of emotion. Cognition and Emotion, 4, 367-390.

Moreno, C. R., Borod, J., Welkowitz, J., & Alpert, M. (1990). Lateralization for the expression and perception of facial emotion as a function of age. Neuropsychologia, 28, 191-209.

Morris, M., Bowers, D., Verfaellie, M., Blonder, L., Cimino, C., Bauer, R. M., & Heilman, K. M. (1992). Lexical denotation and connotation in right and left hemisphere damaged patients. Paper presented at meeting of International Neuropsychological Society, San Diego.

Morris, M. K., Bradley, M., Bowers, D., Lang, P. J., Heilman, K. M. (1991). Valence specific hypoarousal following right temporal lobectomy. Presented at the meeting of the International Neuropsychological Society, San Antonio, TX.

Morrow, L. Vrtunski, P. B., Kim, Y., Boller, F. (1981). Arousal responses to emotional stimuli and laterality of lesions. Neuropsychologia, 19, 65-71.

Notterman, J. M., Schoenfeld, W. N., & Bersh, P. J. (1952). Conditioned heart rate response in human beings during experimental anxiety. Journal of Comparative and Physiological Psychology, 45, 1-8.

OsGood, C. E. (1966). Dimensionality of the semantic space for communication via facial expressions. Scandinavian Journal of Psychology, 7, 1-30.

Papez, J. W. (1937). A proposed mechanism of emotion. Achieves of Neurology and Psychiatry, 38, 725-743.

Patrick, C. J., & Berthot, B. D. (1995). Psychophysiology, 32, 72-80.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1991) Emotion in the Criminal Psychopath: Startle Reflex Modulation. Submitted for publication.

Perret, D., Smith, P., Potter, D., Mislin, A., Head, A., Milner, A. (1984). Neurons responsive to faces in the temporal cortex: Studies of functional organization, sensitivity to identity and relation to perception. Human Neurobiology, 3, 197-204.

Petry, H. M. & Desiderato, O. (1978). Changes in heart rate, muscle activity, and anxiety level following shock threat. Psychophysiology, 15, 398-402.

Reiman,E. M., Fusselman, M. J., Fox, P. T., Raichle, M. E. (1989). Neuroanatomic correlates of anticipatory anxiety. Science, 243, 1071-1074.

Reuter-Lorenz, P. & Davidson, R. J. (1981). differential contributions of the two cerebral hemispheres for perception of happy and sad faces. Neuropsychologia, 19, 609-614.

Richardson, C., Bowers, D., Eyeler, L., Heilman, K. M. (1992). Asymmetrical control of facial emotional expression depends on the means of elicitation. Presented at meeting of International Neuropsychology Society, San Diego.

Rinn, W. E. (1984). The neuropsychology of facial expression: A review of neurological and psychological mechanisms for producing facial expressions. Psychological Bulletin, 95, 52-77.

Robinson, R. G., Kubos, K. L., Starr, L.B., Rao, K., Price, T. R. (1984). Mood disorders in stroke patients. Brain, 107,81-93.

Robinson, R. G. & Price, T. R. (1982). Post-stroke depressive disorders: a follow up study of 103 outpatients. Stroke, 13, 635-641.

Robinson, R. G. & Szetela, B. (1981). Mood change following left hemisphere brain injury. Annuals of Neurology, 9, 447-453.

Ross, E. D. (1981). The aprosodias: Functional-anatomic organization of the affective components of language in the right hemisphere. Annals of Neurology, 38, 561-589.

Ross, E. D., & Mesulam, M. M. (1979). Dominant language functions of the right hemisphere? Achieves of Neurology, 36, 144-148.

Russell (1927/1961). An outline of philosophy. Cleveland: World.

Russell, J. A. & Mehrabian, A. (1977). Evidence for a three-factor theory of emotion, Journal of Research in Personality, 11, 273-294.

Sacheim, H. A., Greenberg, M S., Weiman, A. L., Gur, R. C., Hungerbuhler, J. P. & Geschwind, N. (1982).Hemispheric asymmetry in the expression of positive and negative emotion: Neurologic evidence. Achieves of Neurology, 69, 379-399.

Sackheim, H. A. & Gur, R. C. (1978). Lateral asymmetry in intensity of emotional expression. Neuropsychologia, 16, 473-481.

Schacter, S. & Singer, J. E. (1962) Cognitive, social and physiological determinants of emotional states. Psychological Review, 69, 379-399.

Schlanger, B. B., Schlanger, Pl, & Gerstmann, L. J. (1976). The perception of emotionally toned sentences by right-hemisphere damaged and aphasic subjects. Brain and Language, 3, 396-403.

Scholsberg, H. S. (1941). A scale for the judgement of facial expressions. Journal of Experimental Psychology, 29, 497-510.

Schrant, N. J., Tranel, D., Damasio, H. (1989). The effects of right cerebral lesions on skin conductance response to signal stimuli. Neurology, 39, Supple. 1, 223.

Schwartz, G. E., Ahern G. L., & Brown, S. L. (1979). Lateralized facial muscle responses to positive and negative emotional stimuli. Psychophysiology, 16, 561-571.

Schwartz, G. E., Brown, S. L., Ahern, G. L. (1980). Facial muscle patterning and subjective experience during affective imagery: sex differences. Psychophysiology, 17, 75-82.

Schwartz, G. E., Davidson, R. J., & Maer, F. (1975). Right hemisphere lateralization for emotion in the human brain: Interactions with cognition. Science, 19, 286-288.

Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., Klerman, G. L. (1976a). Facial muscle patterning to affective imagery in depressed and nondepressed subjects. Science, 192, 489-491.

Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., & Klerman, G. L. (1976b). Facial expression and imagery in depression: an electromyographic study. Psychosomatic Medicine, 38, 337-347.

Schwartz, G. E., Weinberger, D. A. (1980). Patterns of emotional responses to affective situations: Relations among happiness,sadness, anger, fear, depression, and anxiety. Motivation and Emotion, 4, 175-191.

Simons, R. F., Ohman, A., & Lang, P. J. (1979). Anticipation and response set: Cortical, cardiac, and electrodermal correlates. Psychophysiology, 16, 222-233.

Sinyor, D., Jacques, P., Kaloupek, D. G., Becker, R., Goldenberg, M., & Coopersmith, H. (1986). Poststroke depression and lesion location. Brain, 109, 537-546.

Sirota, A. D., Schwartz, G. E., & Kristeller, J. L. (1987). Facial muscle activity during induced mood states: differential growth and carry-over of elated versus depressed patterns. Psychophysiology, 24, 691-699.

Slomine, B. S., & Greene, A. F. (1993). Anger imagery and corrugator electromyography. Journal of Psychosomatic Research, 37, 671-676.

Sokolov, E. N. (1963). Perception and the conditioned reflex. Oxford: Pergamon Press.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Starkstein, S. E., Robinson, R. G., & Price, T. R. (1987). Comparison of cortical and subcortical lesions in the production of poststroke mood disorders. Brain, 110, 1045-1059.

Suberi, M., & Mckeever, W. (1977). Differential right hemisphere memory storage of emotional and nonemotional faces. Neuropsychologia, 15, 757-768.

Tassinary, L. G., Cacioppo, J. T., & Geen, T. R. (1989). A psychometric study of surface electrode placements for facial electromyographic recording: The brow and cheek muscle regions. Psychophysiology, 26, 1-16.

Terzian, H. (1964). Behavioral and EEG effects of intracarotid sodium amytal injections. Acta Neurochiurgica, 12, 230-240.

Tomarken, A. J., & Davidson, R. J. (1992). Fear an the face: Differential accessibility of fear and disgust to the face. Cited in R. J. Davidson, Parsing affective space: Perspectives from neuropsychology and psychophysiology. Submitted for publication.

Tomkins, S. (1962). Affect, imagery, and consciousness: The Positive affects, Vol. 1. New York: Springer.

Tomkins, S. (1963). Affect, imagery, and consciousness: The Negative affects, Vol. 2. New York: Springer.

Tranel, D. T. (1983). The effects of monetary incentive and frustrative nonreward on heart rate and electrodermal activi. Psychophysiology, 20, 652-657.

Tranel, D. T. & Damasio, H. (1994). Neuroanatomical correlates of electrodermal skin conductance responses. Psychophysiology, 31, 427-438.

Tucker, D. M. (1981). Lateral brain function, emotion, and conceptualization. Psychological Bulletin, 89, 19-46.

Tucker, D. M., Roth, R. S., Arneson, B. A., & Buckingham, V. (1977). Right-hemisphere activation during stress. Neuropsychologia, 15, 697-700.

Tucker, D. M., Stenslie, C. E., Roth, R. S., & Shearer, S. L. (1981). Right frontal lobe activation and right hemisphere performance decrement during a depressed mood. Achieves of General Psychiatry, 38, 169-174.

Tucker, D. M., Watson, R. T., & Heilman, K. M. (1977). Affective discrimination and evocation in patients with right parietal disease. Neurology, 27, 947-950.

Van Lancker, D., & Sidtis, J. J. (1992). The identification of affective-prosodic stimuli by left and right hemisphere damaged subjects: All errors are not created equal. Journal of Speech and Hearing Research.

Vrana, S. R., Cuthbert, B. N., & Lange, P. J. (1986). Fear imagery and text processing. Psychophysiology, 23, 247-253.

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1989). Processing fearful and neutral sentences: Memory and heart rate change. Cognition and Emotion, 3, 179-195.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology, 54, 1063-1070.

Weinberger, D. A., Schwartz, G. E. (1979). Low-anxious, high-anxious,and repressive coping styles: psychometric patterns and behavioral and physiological responses to stress. Journal of Abnormal Psychology, 88, 369-380.

Weddell, R., Miller, R., Trevarthen, C. (1990). Voluntary emotional facial expressions in patients with focal cerebral lesions. Neuropsychologia, 28, 49-60.

Weintraub, S., Mesulam,, M. M., & Kramer, L. (1981). Disturbances in prosody. Archives of Neurology, 38, 742-744.

Wundt, W. (1896). Grundriss der Psychologie. Translated by C. J. Judd.

Yokoyama, K., Jennings, R., Ackles, P., Hood, P., & Boller, F. (1987). Lack of heart rate changes during an attention demanding task after right hemisphere lesions. Neurology, 37, 748-755.

York, D. J., Cuthbert, B. N., & Lang, P. J. (1989). Imagery of affective sentences: Facial and visceral responses. Psychophysiology, 26, S67.

Zoccolatti, P., Scabini, D., & Violani, C. (1982). Electrodermal responses in patients with unilateral brain damage. Journal of Clinical Neuropsychology, 4, 143-150.

BIOGRAPHICAL SKETCH

Beth S. Slomine was born in Philadelphia, Pennsylvania, on November 19, 1967. She attended the University of Delaware from 1985 to 1989, where she obtained a bachelor's degree with a major in psychology and a minor in biology, graduating magna cum laude. In 1989, Beth began the doctoral program in clinical psychology at University of Florida. After obtaining her master's degree in May, 1992, she began working towards her doctoral degree. She is currently completing her predoctoral internship at the Brockton VA in Massachusetts. After obtaining her Ph.D., Beth will be returning to the Philadelphia area to pursue a postdoctoral fellowhip in geropsychology at the Philadelphia Geriatric Center.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Dawn Bowers, Chair
Associate Professor of Clinical
and Health Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Russell M. Bauer, Cochair
Associate Professor of Clinical
and Health Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Kenneth Heilman
Professor of Clinical and Health
Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Eileen B. Fennell
Professor of Clinical and Health
Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Margaret Bradley
Associate Scientist of Psychology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

P.V. Rao
Professor of Statistics

This dissertation was submitted to the Graduate Faculty of the College of Health Related Professions and to the Graduate School and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

August 1995

Dean, College of Health Related Professions

Dean, Graduate School